MANUAL OF ANESTHESIOLOGY FOR RESIDENTS AND MEDICAL STUDENTS

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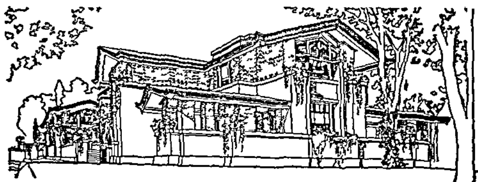
Manual of
ANESTHESIOLOGY

FOR RESIDENTS AND MEDICAL STUDENTS

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INTRODUCTION

ANESTHESIOLOGY is a specialty in medicine requiring the diagnostic skills of the physician and the technical dexterity of the surgeon These twin facets must be developed to the greatest possible degree in the course of the training program But there must be a beginning somewhere It is not sufficient to start technical training without scientific learning Didactic teaching without practical work also omits something of value

After some trial and much error, a reasonably satisfactory solution was found Beginners were taught practical work *in the operating rooms as part of a unit consisting* of the instructor and one or two students They worked together every day as a team for several weeks The confusions of haphazard starts were reduced to a minimum During this period beginners meet thrice weekly to learn 'what every young anesthesiologist should know This manual is their guide The learning process is developed hopefully and possibly even effectively when the manual becomes less important than the reading of original source material and when increased responsibility in the operating room is possible and productive

The manual proved popular with residents in anesthesiology and surgery Medical students wished it whether for knowledge or the passage of examinations was never quite clear In any event, the large demand suggested that its availability in book form would be valuable It is our sincere hope that it will prove useful to beginners everywhere

ACKNOWLEDGMENTS

The manual had many contributors in encouragement, planning, and the writing of earlier versions Gratitude is due them for their unselfish devotion to the preparation of this teaching guide for residents in The Presbyterian Hospital and students in the College of Physicians and Surgeons of Columbia University Some omissions are inevitable and apologies are due them Particular appreciation is freely and gratefully offered Drs Virginia Apgar, M Jack Frumin, Duncan A Holaday, William Howland (now Director of Anesthesiology at Memorial Cancer Center, New York City), Herbert Rackow, and Daniel Tausig (now Director of Anesthesiology, North Shore Hospital, Manhasset, New York)

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CONTENTS

MANUAL OF ANESTHESIOLOGY FOR RESIDENTS AND MEDICAL STUDENTS

I

RECORDS AND CHARTING

THE ANESTHESIA RECORD

THE CAREFUL and frequent recording of data on anesthesia records during the progress of an anesthetic procedure is imperative

1 To insure frequent attention to the patient s vital signs

2 To provide a ready reference of the patient s reaction to anesthesia and operation

3 To establish the sequence of any untoward changes in the vital signs or physiology of the patient should these occur

4 As a source of reference, teaching, and statistical material

5 As a medicolegal record

A copy of the anesthesia record used at the Columbia-Presbyterian Medical Center is provided in this section This consists of an anesthetic chart in duplicate The carbon copy is incorporated in the patient s chart, the original filed in the anesthesia office This is the most important record as it is both a source of material for teaching and research, and a basis for the acquisition of statistical data

Preparation of the chart before starting the case is an important aid in reviewing the patient s condition in detail and recalling the findings acquired during the preanesthetic examination

For purposes of instruction this chart may be divided into three sections

Section I

(Top of the Record)

This section is filled out from the patient s clinical chart The following points are important

Blood Pressure If there are a variety of readings the range is recorded

Hemoglobin and Red Blood Count (Hematocrit if it is available)

Habitus Record as average, thin, obese weight loss *muscular or debilitated*

Teeth Record as bridges, plates, caries, broken, missing, edentulous

Preoperative medication is ordered by the anesthesiologist The amount, time, and effect are recorded for two reasons The patient may be anesthetized again by someone else If the medication was unsatisfactory, it can be changed for the next procedure Secondly, a study of the time, dose, agent, and route of administration will enable one to judge the effects of various medica tions and to use them more intelligently as experience is acquired

Evaluation This consists of the pertinent physical data, unusual laboratory findings, special tests and pre operative complications

Risk This is a standard evaluation and consists of seven physical states as recommended by The American Society of Anesthesiologists [1]

1 A patient who has no organic disease or who has localized disease without systemic disturbance

2 A patient showing a moderate degree of systemic disease

ANESTHESIA RECORD

EVALUATION

MAINTENANCE

AGENTS

MEDICATIONS

DURATION OF ANESTHESIA

DURATION OF OPERATION

3 A patient showing a severe degree of systemic disease

4 A patient with extreme systemic disturbances of imminent danger to life

5 Patients who are to have an emergency operation who would otherwise be regarded as 1 or 2

RECOVE Y ROOM FORM

	MEDICATIONS	ROUTE	Intake	Output	
70 200 1 0 160	1.	Urine			Dressing Draining Yes No Type
70 100 8 60	2.	N. A. tube			
0 20	3.	N. G. tube			
0 S2 EM	4.	T-tube			Oxygen Nasal Tent Mask
	5.				

MEDICATIONS	ANTIBIOTICS	DOSE	TIME	ROUTE
6.	Penic llin			
7.	Crys illi			
8.	Streptomycin			
9.	Aureomycin			
0.	Terramycin			
	Other			

FLUIDS	N/S	5% D/W	BLOOD	P O.	OTHERS
During Oper ion					
In Recovery Room					
T al at Discharge					

ANESTHESIA
E her Cyclopropane E h lene Pentothal
N O/O Caudal Spinal Block Curare
Other
Airway Yes N
Pharyngeal Endotracheal
C ll Pack Tongue sutur
Other

Remarks

Drain

Catheter

ADN ISSION Da Time
D SCHAR E Date Time

SIGNATURE

Condi ion into Recovery Room
Good Fair Poor Terminal
Condition out of Recovery Room
Good Fair Poor Terminal

6 Patients who are to have an emergency operation who would otherwise be classified as 3 or 4

7 The patient who is moribund before emergency operation

Section II

This is the dynamic portion of the chart and during the conduct of the anesthesia gives a minute to minute account of the patient s physiologic status, i e, depth of

anesthesia, blood pressure, pulse rate and respiratory rate, anesthetic agent being administered, fluid therapy

The following diagram (Figure 1) shows an accepted method of recording the agents used and the level of anesthesia in stage 3

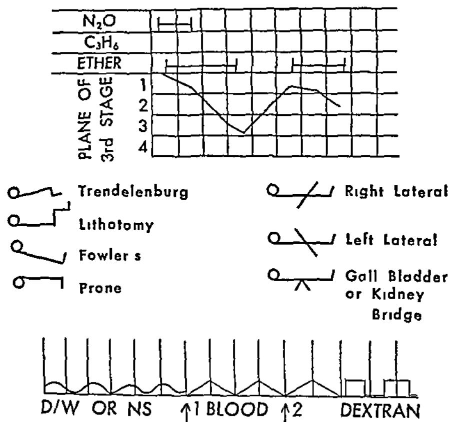


Figure 1

Section III

Agents This consists of the various anesthetic agents used, including general and regional, and the muscular relaxants The primary agent is that which gives the greatest depth or degree of anesthesia The method can vary but a partial list may include

Circle absorption
To and fro absorption
Insufflation
Semi closed
Semi open
Open drop
} + Endotracheal
Spinal
Caudal
Epidural
Block
Topical
Local infiltration

Medications This consists of all medication given during the anesthesia, such as fluids, blood, vasopressors and narcotics

Maintenance Should indicate the type of artificial airway used (metal rubber, plastic), size (Magill or French), and the presence of a cuff, and whether it was inflated The degree of ease or difficulty of exposure of the larynx and any abnormality present should be noted It is necessary to state whether there is trauma associated with the intubation and whether the patient reacted when the tube was inserted into the trachea

If a regional technique is used, specify needle gauge and length and any difficulty of insertion In spinal anesthesia specify the level of the interspace used for lumbar puncture, difficulty and the color of the spinal fluid (clear, bloody etc) With continuous spinal or epidural technique state whether a needle or a catheter is used, the initial dose, the specific agent as well as the initial level of anesthesia, e g , T-4

Postoperative Status Record as good, fair, poor or

terminal The signature in this space is that of the anesthesiologist who administers the anesthetic

Recovery Room Form This record is on the back of the carbon copy which goes with the patient's clinical chart It is an important guide to immediate postoperative care The sections labelled 'fluids,' "anesthesia and 'condition into recovery room" are filled in by the anesthesiologist Under 'fluids' if the unit of fluid is not finished in the operating room it may be listed in this manner

	N/S	*5% D/W*	*Blood*
During operation	500		500
In recovery room			↓

The anesthetic agents and 'techniques used are circled as is the condition into the recovery room

Recovery room nurses record the vital signs at suitable intervals as well as medications given while the patient is in the recovery room

REFERENCE

1 Saklad Meyer Grading of patients for surgical procedures *Anesthesiology* 2 281 284, 1941

II

SIGNS AND STAGES OF GENERAL ANESTHESIA

IN LEARNING to use any drug the physician becomes acquainted with certain signs which indicate whether an inadequate an optimum or a toxic effect is produced by that drug He also notes that the effect of the agent, and the quantity of drug necessary to produce the optimum result varies considerably from patient to patient The anesthesiologist soon observes that these same rules apply to the administration of anesthetic agents Accordingly, in this section the signs indicating only the usual effects of general anesthetics on the patient will be presented and discussed (Figure 2)

These signs have been separated into stages and planes for convenience No one sign alone should be depended upon but rather the situation as a whole should be considered In situations where two signs do not agree as to depth of anesthesia, the more vital sign (respiration pulse, blood pressure) should be relied upon For instance if the eyeballs indicate first plane of the third stage and respiration indicates third plane the anesthesiologist should assume the latter sign to be correct and handle the situation accordingly While administering an anesthetic one is not necessarily striving to reach a certain stage of anesthesia but rather to produce the best operating conditions for the surgeon consistent with the patient's safety As little drug as is necessary should be

	RESPIRATION	PUPILS	EYEBALL ACTIVITY	LOSS OF REFLEX
STAGE I Analgesia				
STAGE II Delerium			++++	
STAGE III Surgical Anesthesia PLANE 1			++ ++ +++ ++ +	A C B D E
PLANE 2				F G
PLANE 3				
PLANE 4				
STAGE IV Respiratory Paralysis				

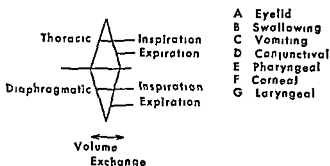


Figure 2 (Modified from Guedel A E *Inhalation Anesthesia* The Macmillan Co 1937)

used to attain these conditions to avoid serious harm to the patient

The signs and stages of anesthesia as described by Guedel[1] apply essentially to the administration of ether

to the non premedicated patient He separates the stages of anesthesia into four subdivisions in the third stage

Stage I Stage of Analgesia (loss or obtundation of pain without loss of consciousness or sense of touch)

This stage exists from the moment of induction to the loss of consciousness Respiration is unchanged The pupils may not change or exhibit moderate reflex dilatation Analgesia is present and amnesia may exist

Stage II Excitement Stage (loss of consciousness to the onset of regular respiration)

In this stage the higher cerebral centers are depressed with loss of inhibition of the secondary centers Excitement and struggling may occur This is one of the danger periods of anesthesia during which vomiting and physical injury may take place Therefore, the anesthesiologist should strive to pass through this stage as quickly as possible In order to obtain a smooth rapid induction, careful attention must be paid to proper and adequate premedication, proper restraint of the extremities, and minimal external sensory stimuli

Respiration is irregular in rate and depth, there may be periods of breath holding The pupils show reflex dilatation Muscular tone is increased in this stage while analgesia and amnesia are present

Stage III Surgical Anesthesia (from the onset of regular respiration to the cessation of spontaneous respiration due to central respiratory paralysis from the action of the anesthetic agent)

Plane 1 Respiration is regular with an increase in tidal volume There is nystagmus and the pupils are constricted There is loss of the vomiting, swallowing, lid, and pharyngeal reflexes Small muscle tone is lost

Plane 2 Respiration is regular but shallower The eyeballs are fixed (central) and the pupil is in mid dila-

tation There is a loss of the corneal, visceral, laryngeal, and cough reflexes The large muscles are beginning to relax in this plane

PLANE 3 This plane is entered when there is the beginning of progressive intercostal lag in which diaphragmatic movement precedes the action of the intercostals It ends with the cessation of intercostal movement The pupil is moderately dilated and all muscle tone (except diaphragmatic) is lost

PLANE 4 Respiration shows complete intercostal paralysis and breathing is diaphragmatic Inspiration is short and gasping with retraction of the intercostal spaces The pupils show paralytic dilatation

Stage IV This stage begins with respiratory paralysis *and progresses quickly to circulatory failure*

Pertinent Notes on the Signs of Anesthesia

The eyelash reflex is related to the eyelid reflex If the eyelash is stroked or the eyelid opened, the patient will actively constrict the orbicularis oculi muscle in the second stage In the third stage the eyelid will close passively or remain open, and stroking the eyelash will elicit no response

Since swallowing will occur before vomiting when the patient is becoming 'light, the continuous presence of the anesthesiologist s hand on the patient s chin will aid in detecting signs of swallowing and permit him to reverse the depth of anesthesia before vomiting occurs

Pupillary size is frequently influenced by pre anesthetic drugs and is often not a reliable sign of anesthetic depth However, a widely dilated pupil with little or no iris visible should always cause concern because it may be the result of hypoxia or excessively deep anesthesia

The most reliable guide to the depth of anesthesia is

respiration if the airway is patent If the airway is obstructed the chest will retract on inspiration even in light planes Use of premedicant drugs may produce respiratory depression in some patients The use of cyclopropane or thiobarbiturates (which unlike ether depress respiration even in light planes) may result in early hypoventilation Assisting respiration under these circumstances is essential to the maintenance of normal gas exchange Pupillary reactions are frequently altered with these agents

An interesting and very useful discussion of the clinical signs of anesthesia was presented by T A B Harris[2] He states that these signs are a consequence of depression in a standard sequence of specific areas of functional activity of the brain by the anesthetic agent as shown below

Standard Sequence of Biological Response			*Guedel's Classification*	
Depression of higher centers (cortex)	Amnesia		Stage I	
	Cooperative stupor			
	Non Cooperative stupor		Stage II	
	Anesthetic sleep		Stage III	Plane 1
Depression of areas of sensory coordination	Loss of ability to react to external stimulus		Stage III	Plane 2
Depression of areas of motor coordination	Loss of muscle tone in	Sm muscle groups	Stage III	Plane 3
		Lge muscle groups		
		Intercostals	Stage III	Plane 4
Depression of vital medullary centers	Failure of respiratory center			
	Failure of vasomotor center			
	Failure of cardiac center		Stage IV	

When the state of anesthetic sleep is reached physiological protective reflexes of the pharynx larynx and

trachea (cough reflex) may still be present External stimulation can still produce these reflex responses and be a danger to the patient It is not, according to Harris, until the patient has reached the state of depression of the areas of sensory coordination (plane 2) that he is freed from the deleterious consequences of the two uncontrollable variables, emotion and external stimulation

In summary, it must be remembered while judging the depth of anesthesia, that the anesthetized patient is not one organ system but an entity If the anesthesiologist is not sure whether the patient is too deep or too light, always consider the patient too deep and lighten the anesthesia This philosophy is preferred to pushing more drug which may result in overdosage and death If any detrimental change in the vital signs (respiration, pulse or blood pressure) occurs, immediate steps to lighten the depth of anesthesia should be taken in an attempt to remedy the situation Only experience with various types of agents in different types of patients will enable the neophyte anesthesiologist to gauge more accurately the depth of anesthesia He must maintain close and continuous vigilance during the administration of the anesthetic to be able to judge the reason for even small changes in signs

REFERENCES

1 Guedel A E *Inhalation Anesthesia* New York Mac millan 1937

2 Harris T A B *Mode of Action of Anesthetics* Edin burgh Livingstone 1951

III

PREMEDICATION

THE MAIN purposes of premedication are

1 *The diminution of anxiety before the administration of anesthesia*

2 The facilitation of a more tranquil induction and maintenance of general anesthesia

3 The prevention of undesirable side reactions to the anesthetic agents

All of these can be summarized by stating that premedicant drugs lower the patient s alertness and his tissues requirement for oxygen

Certain factors will influence basal metabolic rate or oxygen requirement and these influences the dose and type of drugs for premedication

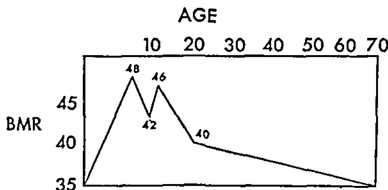


Figure 3 (From Guedel A E *Inhalation Anesthesia* The Macmillan Co 1937)

1 *Age (Figure 3)* BMR varies with age until 20 years At birth it is about 35 Cal /hr /m ^{2} of body surface By one year it increases to 40 Cal, and at six years it reaches its highest level of 48 Cal Then follows a moderate drop to 42 at 10 or 11, and a sudden rise at puberty to 46 From the age of 12 or 13 the basal metabolic rate steadily goes down at a faster rate until it reaches 40 at 20 years and then very gradually to 35 at age 70

2 *Fever* Each degree of fever will raise the BMR about 7% It must be kept in mind that this refers to acute rises in temperature because a patient who has had a long bout of pyrexia will also be toxic" and therefore have a diminished reflex irritability

3 *Toxemia, Hemorrhage and Shock* Toxic or debilitated patients require less preoperative medication This also applies to patients who have had marked blood loss or are in shock

4 *Pain* The presence of pain will raise reflex irritability In these patients the use of a narcotic in the premedication to relieve pain is desirable Barbiturates have little or no analgesic power and tend to make patients in severe pain uncooperative

5 *Endocrine Disturbances* Patients with hyperthyroidism may have a markedly elevated BMR while those with hypothyroid function have a low BMR Patients with adrenal cortical insufficiency and myasthenia gravis require less premedication

6 *Body Build* The muscular robust individual will need more premedication to ensure a tranquil anesthesia while the obese and phlegmatic individual needs less

7 *Alcoholism* The chronic alcoholic is resistant to anesthetic agents Even the use of heavy preoperative medication may be unsuccessful in preventing a stormy induction and maintenance No easy solution is available

for these patients The chronic alcoholic does well with regional anesthesia, the acute alcoholic is usually too un cooperative

8 Emotional Disturbances Many patients have some fear and apprehension of anesthesia and surgery The greater this fear the greater the need for premedication All degrees will be encountered depending on personality and previous anesthetic experiences Much can be done to diminish these apprehensions not only bv adequate doses of the proper drugs, but also by preoperative visits by the anesthesiologist to acquaint the patient with what will happen the day of the operation and to minimize the anticipation of an ordeal that may have developed in his imagination The importance of this preoperative visit must be elaborated in more detail because it is vital in the practice of anesthesiology

The administration of anesthesia must be preceded by a study of the needs of the patient This study requires a careful consideration of all the available information *Since these data are collected usually by people* not primarily interested in the anesthesiological point of view, they must be supplemented by further history or physical examination by the anesthesiologist, and on occa sion by supplemental laboratory investigations which he will request These studies enable him to make an evaluation of the patient s needs at the preoperative visit This visit is not solely to assess physical needs It also establishes a personal relationship between the physician and patient which is essential to the successful practice of medicine in any of its specialties The patient should be told in advance what to expect in the immediate postoperative period Knowledge of the facts is of great help in reducing fear This is also the time to find out whether

the patient has had any previous experience with anesthesia Particular attention is directed to a history of cough, asthma, allergy, impaired exercise tolerance, the state of dental health, the consumption of alcohol, and allergy to drugs Estimates should be made of the patient s state of hydration, the presence of cyanosis, dental repair, nasal obstruction tested in both nostrils independently, the mobility of joints which may be flexed during the operation, the position of the trachea, cardiac rhythm, and ventilatory excursion of the chest It should be remembered that the anesthesiologist is often the last physican to see the patient on the night before the operation and he should attempt to complete his interview with the patient in a restful and optimistic frame of mind Explanations about the details of anesthetic procedures are kept to a minimum Mention should be made in simple terms of the procedures the patient will undergo while still conscious He should also be told where he will be when he recovers consciousness After the interview, a summary of the findings from an anesthesiological point of view should be written on the chart The general plan for anesthetic management is summarized in the progress note Details are unnecessary On the operative day some patients will arrive in the anesthesia room in a state of apprehension in spite of the premedication Everything should be in readiness therefore, so that the period of suspense can be kept to a minimum and the anesthesia begun as soon as possible filling in the time by appropriately reassuring conversation

The number of postoperative visits that should be made to the patient depend on the length of the illness and the nature of the surgery It should, in any case, never be less than one and should be made daily until the patient s recovery is assured These visits usually

aid the surgeon and the internist in the postoperative care of the patient and help to reassure the patient that he is receiving maximum care from all his physicians

Drugs Used for Premedication The drugs used for premedication are sedatives, hypnotics and drying agents

I NARCOTICS AND HYPNOTICS These are used to diminish alertness, produce tranquility and depress anxiety They are also important in relieving pain if it is present The narcotics usually employed are morphine or Demerol® (meperidine) and the usual hypnotics used are barbiturates (pentobarbital and secobarbital)

Morphine This drug is a phenanthrene derivative of opium Its most important desirable action is on the central nervous system It is a good analgesic It raises the pain threshold and abolishes hunger, thirst fatigue and anxiety It produces a feeling of well being (euphoria) and tends to relax the patient In the usual premedicant doses the patient may get drowsy and fall asleep but can be awakened easily

The effect of morphine on respiration is depression Overdose will cause slow deep respirations and may lead to shallow slow respirations with asphyxia Circulatory depression manifested by falls in arterial blood pressure may be produced either as a direct effect of the drug or as a consequence of the respiratory depression Therapy of overdose includes artificial ventilation with 100% oxygen, intravenous injection of 5 to 10 mgm doses of an antagonist *nalorphine* (*n allyl normorphine*) and possibly vasopressors Overdose complications are not frequent in incidence

The time interval between the injection of morphine and the induction of anesthesia should be sufficiently long so that the peak of respiratory depression has already been passed The time of effect depends upon the route

used If given subcutaneously, about one hour is required to produce the maximum depression of respiration although the analgesic effect will be evidenced in 15 minutes When given intramuscularly maximum depression occurs in 30 minutes, intravenously 7 to 10 minutes When premedication is given intravenously only one-half to two-thirds of the intramuscular dose is needed The average dose of morphine for premedication of healthy adults (varies considerably with the patient s age, condition, weight) is from 8 to 15 mgms

Other effects of morphine

Nausea and emesis are common side effects probably because of stimulation of the vomiting center Morphine causes spasm of all segments of the gastrointestinal tract, especially the sphincters and is constipating This effect may contribute to postoperative ileus Morphine also can cause spasm of the bronchioles The cough reflex is markedly reduced centrally and is advantageous in allowing for a smooth anesthetic induction It is avoided in patients with a history of biliary colic and bronchial asthma

Demerol® (Meperidine) This is a synthetic compound which has about one-tenth the potency of morphine Its psychic effect and the respiratory and circulatory depressant responses do not differ remarkably from morphine when given in equipotent doses Nausea and vomiting may occur It is antispasmodic to the gastrointestinal tract and bronchioles and has some drying effect on respiratory tract secretions The cough reflex is depressed The average dose is 50 to 100 mgms and overdose is antagonized by nalorphine

Barbiturates These drugs reduce apprehension and fear However, their important action is the production of drowsiness and sleep These drugs are not analgesic A patient who has received a barbiturate and is exposed

to painful stimuli may become restless and uncooperative These drugs are best used in conjunction with a narcotic or during the morning for afternoon operations Over dose causes respiratory depression with rapid shallow respirations N allyl normorphine does *not* antagonize the barbiturates Assisted breathing with oxygen through an open airway is indicated

The usual adult dose is 100 mgms of pentobarbital or secobarbital by mouth about 1½ hours before anes thesia is to begin If morphine is also used the barbiturate is given about 30 minutes before the morphine Seconal® is also available for intramuscular use The hypnotic effect of the barbiturates is especially useful in sedating the patient during the operative procedure if regional meth ods of anesthesia are used

Since overmedication with either the narcotics or bar biturates will produce respiratory depression, the estab lishment of deeper levels of anesthesia with inhalation agents may be slowed There is often technical difficulty caused by the development of apnea before satisfactory depth of anesthesia is obtained

II BELLADONNA DERIVATIVES These drugs are used for premedication to reduce secretions in the respiratory tract and partially depress vagal reflexes

Atropine and scopolamine are the usual drugs used for this purpose Scopolamine counteracts somewhat the respiratory depression of the narcotics and also has the *added advantage of causing amnesia Sweating is in*hibited by these drugs The heat regulating mechanism of the body can be disturbed and heat retention ensue Since both drugs are vagolytic, large doses (1 5 2 0 mgms) will produce tachycardia The usual adult dose is 0 3 to 0 5 mgms given with the narcotic via the same parenteral route

III THORAZINE® (CHLORPROMAZINE) This is a relatively new drug which can be used preoperatively It will diminish anxiety and apprehension and relax the patient Since it potentiates the effects of narcotic and anesthetic drugs, the amounts of these agents required to induce and maintain general anesthesia are lessened Chlorpromazine also has an antiemetic effect Its greatest disadvantage is hypotension The usual adult dose is 12 5 to 25 mgms given intramuscularly one to two hours before anesthesia It can also be given orally in doses up to 50 mgms about 2 hours before the induction of anesthesia Since chlorpromazine potentiates the narcotics, the doses of the latter can be reduced by one half the amount normally used

IV

NEURAL AND CHEMICAL CONTROL OF RESPIRATION

NEURAL CONTROL OF RESPIRATION

THE NEURAL CENTERS for the control of respiration are located in the brain stem The inspiratory and expiratory centers as described by Pitts *et al*[1] are localized in the medial and lateral reticular formation of the medulla over the rostral four fifths of the inferior olivary nuclei The expiratory center is situated caudal to the inspiratory center From these centers rhythmic impulses are discharged through descending pathways to the anterior horn motor neurones of the cervical and dorsal spinal cord and then through the phrenic and intercostal nerves to the respiratory muscles

The origin of the respiratory rhythm has been the subject of investigation for many years The current theory of Pitts holds that the medullary inspiratory center does not have any inherent rhythmicity but is tonically active The respiratory rhythm is the result of periodic inhibition of the inspiratory center by impulses from the pneumotaxic center and vagal afferents However, experiments done in our laboratory[2] and by others in the past few years demonstrated that the central neural regulation of the respiratory movements is indeed more complicated than this current hypothesis The center complex

consists of several neural structures located throughout the reticular formation of the pons and medulla (Figure 4) The anatomical localization and physiological functions of the respiratory center complex as we understand them at the present can be summarized briefly

The medullary inspiratory center is not tonically active Under normal conditions the medullary respiratory center is under the influence of pontile respiratory centers and is probably a common motor pathway for higher neural structures In the pons an apneustic center is located in the lateral reticular formation in its caudal portion and a pneumotaxic center is located in the dorsolateral reticular formation at the level of the isthmus The normal respiratory movement is the result of interaction of the apneustic center with the vagal afferents and the pneumotaxic center During inspiration as the lungs are inflated the pulmonary stretch receptors are stimulated Afferent impulses ascend through the vagus nerves to the brain stem and exert inhibitory influence to stop inspiration and start expiration (the Hering Breuer reflex) The active apneustic center also sends excitatory impulses to the pneumotaxic center Either one of these two feedback mechanisms is capable of giving rhythmical respiration If both of these mechanisms are removed the uninhibited apneustic center, because of its tonic activity, will give prolonged inspiratory spasm, apneusis

The respiratory movements are also under the influence of cortical activity, the hypothalamic centers and various afferent impulses Breath holding, voluntary hyperventilation, sneezing and coughing occurs in response to foreign bodies in the air passage, hyperpnea and tachypnea may follow noxious somatic stimulation, and hiccough may result from diaphragmatic irritation or other stimulation

CHEMICAL REGULATION OF PULMONARY VENTILATION

Pulmonary ventilation is regulated by the activity of the respiratory center to achieve adequate gas exchange for the absorption of oxygen and the elimination of carbon dioxide Chemosensitive cells are present in the center as well as in the peripheral receptors of the carotid and aortic bodies Small changes in the tensions of respiratory gases and H-ion concentration in arterial blood are detected by these chemoreceptors and pulmonary ventilation is adjusted to bring the blood gas tension and H ion concentration toward normal levels

1 Oxygen

Normal arterial oxygen tension (pO_2) is about 100 mm Hg An increase in arterial tension has an insignificant effect on ventilation A decrease of the oxygen tension stimulates ventilation markedly through the carotid body mechanism provided that the function of the respiratory center is not depressed by anoxia or anesthesia For practical purposes, the direct effect of anoxia on the respiratory center is depression

2 Carbon Dioxide

The normal arterial carbon dioxide tension (pCO_2) is about 40 mm Hg A slight change in the arterial pCO_2 is reflected promptly on the pulmonary ventilation It is generally agreed that carbon dioxide has a specific stimulating action on the respiratory center neurones although changes in intracellular H ion concentration associated with pCO_2 changes also play a part

a A decrease in arterial pCO_2 results in a decrease in ventilation Voluntary hyperventilation is followed by a period of apnea from hypocapnea Respiration

Figure 4 Schematic diagram of interrelationship of components of respiratory center complex Influence of other neural impulses and chemical regulation

resumes when the arterial pCO_2 is back to the threshold level again

b An increase in pCO_2 results in augmentation of ventilation Both the rate and depth of respiration are increased In normal unanesthetized subjects, 5% carbon dioxide in the inspired air increases the ventilatory vol-

ume fourfold, 10% carbon dioxide tenfold (the maximal stimulating concentration with carbon dioxide) Carbon dioxide in concentrations higher than 10% is intolerable to the conscious subject, 30% CO_2 is anesthetic and concentrations higher than 40% are depressant to the respiratory center

Under normal conditions carbon dioxide has a tonic stimulating effect on the carotid body as well as on the center itself This is supported by the fact that denervation of the carotid body is followed by an increase in alveolar pCO_2, indicating that the pulmonary ventilation is decreased and the equilibrium is established at a higher level With a higher concentration of carbon dioxide its effect is predominantly central, as denervation of the carotid body under this condition does not alter the respiratory response It is possible that the high carbon dioxide cencentration depresses the central synaptic transmission so that an increase in the activity of the carotid body is not apparent

c *H-ion Concentration* Although the respiratory responses to changes in H ion concentration are in the same direction as the carbon dioxide changes, the response is not as profound Hyperpnea from metabolic acidosis is accompanied by a decrease in arterial pCO_2

COMMON DERANGEMENTS OF REGULATION OF RESPIRATION DURING ANESTHESIA

1 Drugs

Narcotics, barbiturates and most general anesthetic agents (especially cyclopropane) depress the respiratory center The depressant actions of premedicants and general anesthetics are additive so that the resultant depression is the sum of action of each agent The threshold of the respiratory center to carbon dioxide is elevated Ven-

tilation is always decreased and equilibrium is established at a higher level of carbon dioxide tension Frequently, as a result of decreased ventilation, the arterial oxygen tension falls When the arterial oxygen tension falls the carotid body is stimulated and ventilation is maintained mostly through the carotid body reflex mechanism Under these conditions, the administration of gases with a high tension of oxygen will remove the anoxic drive and result in apnea (oxygen apnea) However, oxygen should not be withheld but should be administered with assisted respiration until the patient can spontaneously ventilate adequately

When the central depression is severe there is no ventilatory effort even in the presence of hypercapnea and anoxia No respiratory stimulant can take the place of artificial ventilation It is the only means of maintaining adequate gas exchange until the depressing agent is eliminated and the regulatory mechanisms recover

A depressed center is not stimulated by carbon dioxide Furthermore, it is depressed by the increase of pCO_2 (carbon dioxide reversal) Therefore, there is no physiological rationale in treating patients with depressed respiration with carbon dioxide and the practice is dangerous

2 "Controlled" Respiration

When apnea is produced and the anesthesiologist assumes the responsibility of the patient s ventilation, the condition is defined as controlled" respiration It can be produced by

a Depressing the center with drugs, thus raising the carbon dioxide threshold of the center

b Over-ventilation to reduce the arterial pCO_2 to values below that of threshold

c Paralysis of respiratory muscles with relaxants or deep anesthesia

d Forceful inflation of the lungs to stimulate the vagal pulmonary stretch receptors and thus to inhibit inspiratory efforts — Hering Breuer reflex (It is unlikely that this factor alone can produce apnea)

Controlled' respiration is useful to obtain better muscular relaxation and to provide a quiet operating field, especially for operations in the thorax and upper abdominal cavity

Controlled respiration should always be adequate to maintain respiratory gases in the arterial blood close to normal values The recommended rate of breathing is 15 to 20 per minute (adult) Each respiratory cycle should be divided so that one third of the cycle is for inspiration and two thirds for passive expiration As the intrapleural and intrapulmonic pressures are reversed from negative to positive values during the inspiratory phase and remain positive during expiration, excessive and prolonged controlled respiration will impede the venous return to the heart This circulatory effect of pressure breathing may be deleterious to those patients whose circulatory status is poor

REFERENCES

1 Pitts R F Magoun H W and Ranson S W Localization of the medullary respiratory centers in the cat *Am J Physiol*, *126* 673-689 1939

2 Ngai S H and Wang S C Pneumotoxic center in the cat effects of its stimulation and destruction *Am J Physiol*, *171* 752 753 1952

V

PULMONARY FUNCTION

To FACILITATE subsequent discussion, definitions of commonly used terms in the study of pulmonary function are briefly outlined

Tidal Volume, TV The volume of gas moving in and out of the respiratory apparatus during quiet inspiration and expiration

Vital Capacity, VC The maximum volume of gas that can be expelled from the lung by forceful effort following a maximal inspiration

Inspiratory Capacity, IC (complemental air) The maximum volume of gas that can be inspired from the resting expiratory level

Inspiratory Reserve Volume, IR The maximum amount of gas that can be inspired from the end inspiratory position

Expiratory Reserve Volume, ER (supplemental air) The maximum volume of gas that can be expelled from the resting end-expiratory position In function studies the resting expiratory position is always used

Functional Residual Capacity, FRC (functional residual air) The volume of gas remaining in the lungs in the resting end expiratory position

Residual Volume, RV (residual air) The volume of gas remaining in the lung at the end of maximal expiration

Total Lung Capacity, TC The sum of vital capacity

and residual volume, the maximum amount of gas that can be contained in the lung when it is fully expanded

Resting Ventilation The amount of gas moved in and out of the lungs at rest in unit time — usually expressed as liters per minute

Walking Ventilation The amount of gas moved in and out of the lungs in unit time when the individual is walking at a slow, even pace, in liters per minute

Maximal Breathing Capacity, MBC The maximal volume of gas that can be ventilated by the individual in unit time in liters per minute

Breathing Reserve The maximal breathing capacity minus the resting ventilation in liters per minute

Alveolar Ventilation, V_A The volume of inspired air that enters the alveoli and participates in active gas exchange

Dead Space Ventilation, V_D The volume of inspired air that does not participate in active gas exchange It is

TABLE I

NORMAL VALUES

(Liters 37 C saturated with water vapor)

	Young Males	*Older Males*	*Females*
Tidal volume TV	0 5		
Inspiratory capacity IC	3 8	3 4	2 4
Expiratory reserve volume ER	1 0	0 7	0 7
Vital capacity VC	4 8	4 1	3 1
Residual volume RV	1 2	1 3	1 1
Total lung capacity TC	6 0	5 4	4 2
Functional residual capacity FRC	2 2	2 0	1 8
RV/TC x 100	19 8	24 5	25 9
Maximal breathing capacity	120/min	90/min	90/min
Breathing reserve/MBC x 100		95 9% at rest	
Walking ventilation/MBC		< 0 3	
Dead space ventilation V_D	0 5		0 12
V_D/TV x 100		<30	
Venous admixture/cardiac output x 100		< 6	
O_2 Diffusion capacity		>15 cc /min /mm Hg pressure gradient	

the sum of the anatomical dead space and the amount of air ventilating the alveoli which are not perfused with blood

Oxygen Diffusion Capacity, D_{O_2} Amount of oxygen diffused across the alveolar capillary membrane in unit time per unit pressure gradient, as cc /min /mm Hg

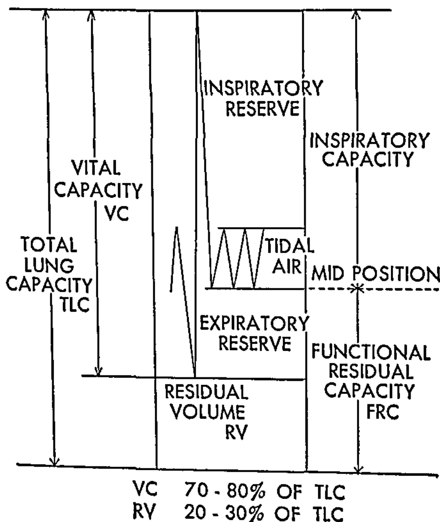


Figure 5

NORMAL PULMONARY FUNCTION

The anatomical and physiological aspects of pulmonary function can be divided into three categories

I Pulmonary Ventilation Involving the mass movement of gas in and out of the lungs which is controlled by the action of the chest The rhythmic movements of this bellows carry on the work of breathing and in so doing have to overcome the elastic resistance of the lung and thorax and the resistance of the gas moving through the air passages To provide normal ventilation the integrity of structures and function of the respiratory apparatus is necessary

A Bony framework of the thoracic cage and adjacent parts
B Respiratory muscles
C Normal structure and elasticity of lungs, pleura and mediastinal contents
D Patent airway
E Normal respiratory stimuli — arterial pCO_2 and pH

II Respiratory Gas Exchange, Alveolar Respiratory Function This is particularly concerned with the effective distribution of inspired gas to the functional alveoli and with the gas exchange across the alveolar-capillary membrane This is the final purpose of pulmonary ventilation The effective tidal air must be as evenly distributed as possible to actively perfused alveoli Under normal conditions the inspired gas is well distributed and most ventilated alveoli are perfused with blood The effectiveness of inspired air in displacing vitiated air in the alveoli depends on its volume and the volume of residual air Shallow breathing is relatively ineffective

The rate at which gas diffusion takes place is con

trolled by the physical properties of the alveolar-capillary membrane and its total area In normal lungs there is no barrier

III Pulmonary Blood Flow This is concerned with the capillary blood perfusion, total blood flow and pulmonary arterial pressure Intact function of the heart is essential

COMMON PULMONARY DYSFUNCTIONS OF IMPORTANCE IN ANESTHESIA

Anesthetic pulmonary dysfunctions can be classified into four categories Each condition can exist alone or in any combination

I Reduction of Ventilatory Capacity Because of the reduced ventilatory capacity the individual tends to have dyspnea The common pulmonary function tests for the detection and estimation of the extent of this disturbance are the measurements of resting ventilation, walking ventilation and maximum breathing capacity The breathing reserve ratio (breathing reserve/maximum breathing capacity)and the walking ventilation/maximum breathing capacity ratio offer good indices of the patient s ability to stand stress The normal value of breathing reserve ratio is 95 9 Individuals with a breathing reserve ratio of 80 or less will have dyspnea during exercise of two minutes duration Dyspnea at rest will occur in patients with a breathing reserve ratio of 60 70 The normal values for walking ventilation/maximum breathing capacity are 0 3 or less Patients with a ratio of 0 5 will have severe dypsnea This ratio is of particular value in evaluating patients for intrathoracic procedures Patients with a ratio of 0 35 are considered borderline risks and patients with a ratio of 0 5 are extreme risks Simple evaluations of patients ventilation capacity and breathing reserve can

be easily accomplished by inquiry into the patient's history as to *how much he can do without dyspnea*

Conditions with reduced ventilatory capacity are

1 Restriction in pulmonary expansion and contraction, from pulmonary fibrosis, silicosis, pleural thickening, pleural effusion, pneumothorax, and defects and inadequacies of the thoracic cage, e g, rickets, kyphoscoliosis and pneumonectomy

2 Paralysis or weakness of respiratory muscles due to poliomyelitis, myasthenia gravis and other diseases

3 Obstruction of air passages at any point from nose to alveoli

4 Pulmonary emphysema

II Excessive Dead Space Ventilation Excessive dead space ventilation reduces the effective alveolar ventilation The ratio of the dead space ventilation to the tidal volume determines the effective alveolar ventilation Even normal ventilation can result in inadequate alveolar ventilation if shallow breathing reduces tidal volume significantly In normal individuals the value of dead space ventilation/tidal volume should be less than 0 3 Some conditions with increased dead space ventilation are

1 Air cysts in the lung and emphysematous bulli
2 Multiple pulmonary embolization
3 Pulmonary ventilation without perfusion

All these conditions contribute to an increase in the amount of air which is being ventilated in the alveoli but is not brought into contact with the blood for gas exchange

III Excessive Venous Admixture In this condition the blood is perfused through areas which are not being

ventilated and therefore does not contribute to gas exchange In normal individuals the percentage of venous admixture in respect to the right ventricular output (venous admixture/cardiac output × 100) is less than 6 Conditions with excessive venous admixture are

1 Consolidation of the lung tissues in atelectasis and pneumonitis

2 Obstruction of the main bronchus or its branches

3 Large arterio venous shunt in the pulmonary circulation which is rare

IV Reduction in Oxygen Diffusing Capacity of the Alveolar Capillary Membrane The normal oxygen diffusion capacity should be greater than 15 cc /min /mm Hg of pressure gradient It can be reduced when the alveolar wall is separated from the pulmonary capillary wall by disease processes Reduction of diffusing capacity occurs in

1 Pulmonary edema

2 Interstitial pneumonitis

3 Pulmonary fibrosis, granulomata and miliary infiltration of the lung

DISTURBANCES OF PULMONARY FUNCTION DURING ANESTHESIA AND OPERATION

Associated with the induction of anesthesia, positioning of the patient and the performance of surgical procedures pulmonary function can be disturbed in many ways The same classification of pulmonary dysfunction can be followed

I Reduced Ventilatory Capacity

1 Depressed respiratory centers, lack of adequate stimulus to activate the rhythmical movements of respira-

tory muscles This is discussed in the section of neural control of respiration (page 28)

2 Paralysis or weakness of respiratory muscles caused by anesthetic agents or muscle relaxants

3 Limitation of thoracic and diaphragmatic movements by extreme postures, e g, steep Trendelenburg and kidney position, by pressure of heavy drapes, instruments or members of the surgical team leaning on the chest, excessive packing of abdominal contents against the diaphragm open thorax and tight strapping and bandaging of the thorax and upper abdomen postoperatively

4 Obstruction of airway Discussed in the section of complications during general anesthesia (page 99)

5 Resistance to gas movement in the anesthetic apparatus There is no significant difference between the resistance in a circle or a to and fro system Open drop technic has the least resistance

Airway obstruction should be corrected *promptly* Mechanical limitation of respiratory movements should be avoided and *at all times the ventilatory effort of the patient should be assisted during general anesthesia*

II Excessive Dead Space Ventilation

1 If the tidal volume is reduced the dead space ventilation becomes greater proportionately and alveolar ventilation can be inadequate

2 The dead space can be increased significantly just by placing the anesthesia mask over the patient's face In a circle carbon dioxide absorption system the dead space in the machine is the volume of the mask and the chimney Y proximal to the point of bifurcation Beyond this point the gas moves only in one direction so that the expiratory tube does not contribute to the dead space In a to and fro carbon dioxide absorption system all the

space proximal to the lime in the canister constitutes the dead space This is increased as the proximal portion of the lime in the canister is exhausted during its usage A medium sized adult anesthesia mask has a volume of 120 cc A child s mask has a volume of 40 cc Considering the normal dead space ventilation in children the addition of this amount can prove to be too much of a burden for efficient alveolar ventilation Endotracheal airways eliminate the dead space of the mask and also the anatomical dead space of the nose, mouth and pharynx Again, because of this mechanical factor *the respiration should be assisted at all times even when the tidal volume appears normal*

3 During pulmonary resection, if the pulmonary artery or vein is ligated before the bronchus is occluded this portion of lung is ventilated but not perfused The dead space ventilation is therefore increased until the bronchus is occluded

III Excessive Venous Admixture

This can occur during anesthesia and operation because of

1 Obstruction of the main bronchus or its branches by secretions, blood, or foreign bodies Ventilation of the lung distal to the obstruction is impaired

2 Endobronchial intubation, the contralateral lung is not ventilated and is perfused

3 Atelectasis

4 During pulmonary resection when the bronchus is occluded before the pulmonary vessels are ligated

IV Reduction of the Oxygen Diffusion Capacity

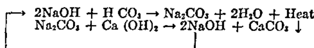
Pulmonary edema can develop during anesthesia from

airway obstruction or overloading of the circulation by excessive fluid infusion or excessive transfusion

Some of these disturbances are inherent to the state of anesthetization, positioning of the patient and the surgical procedures Their existence should be fully appre ciated Conscientious effort should be made to compen sate for these disturbances Some of the other difficulties need not occur if the anesthesiologist is diligent and care ful in the management of anesthesia Complications should be treated promptly

CARBON DIOXIDE ABSORPTION

In a closed rebreathing system, whether it be circle or 'to and fro the carbon dioxide constantly eliminated by the patient must be removed efficiently The most com monly used carbon dioxide absorbents are soda lime and baralyme There are two types of soda lime the wet or high moisture type, containing 14 to 19% water and the dry or low moisture type with 4 to 5% water A mixture of sodium hydroxide (5%) and calcium hydroxide (95%) performs the bulk of the work However, this mixture crumbles easily and it is hardened with silica The commonly used silica is Kieselguhr a diatomaceous earth The reaction of the absorption is as follows



During this reaction heat is liberated at the rate of 13 700 calories for each molecule of water formed

Baralyme is a mixture of 20% barium hydroxide octahydrate and 80% calcium hydroxide This mixture does not require silica as hardening agent The barium hydroxide assumes the role of the activator like sodium

hydroxide in soda lime Soda lime can be reactivated for a while, if allowed to rest' when it becomes exhausted (see equation on page 40) No regeneration of activity occurs with the baralyme

It has been shown that the optimum size and shape of an adult canister is a cylinder with a diameter of 8 cm and a length of 13 cm The volume of such a canister is 650 cc and holds 500 to 550 gms of soda lime or baralyme The dead space is approximately 125 to 150 cc The dead space plus the granular space is 400 to 425 cc Thus the tidal volume must equal or exceed this figure for maximum efficiency

The temperature developed in the canister during absorption may exceed 60 C At the mask the temperature varies between 39 to 41 C in the to and fro system and 32 to 33 C in the circle system

VI

MECHANISM OF TRANSPORT OF THE RESPIRATORY GASES

I OXYGEN TRANSPORT

OXYGEN is transported by the blood by two mechanisms in combination with hemoglobin and in simple solution

A Hemoglobin This blood protein is carried in the red blood cell and its prime function is that of oxygen transport Hemoglobin has the capacity of combining very readily with oxygen under certain conditions and giving up this oxygen under other conditions One mole of oxygen combines reversibly with one mole of hemo globin according to the equation $O_2 + Hb \rightleftharpoons HbO_2$ The relative amounts of reduced hemoglobin (Hb) and oxyhemoglobin (HbO_2) depend to the greatest extent on two factors

1 The pressure of oxygen in contact with the hemoglobin which tends to drive the reaction to the right when the oxygen tension is high, and to the left when the oxygen tension is low

2 The acidity or amount of carbon dioxide present—a high CO_2 driving the reaction to the left, and a low CO_2 having a reverse effect

In the lungs the O_2 tension in the alveoli is higher and the CO_2 tension is lower than that in the venous blood CO_2 diffuses out into the alveoli making the blood less acid This causes a greater affinity of hemoglobin for

oxygen which is also present at a higher tension in the alveoli than in blood O_2 readily combines with hemoglobin for transport to the tissues because of these two factors

TABLE II

COMPOSITION OF INSPIRED AND EXPIRED AIR AND GAS CONTENT OF ARTERIAL AND VENOUS BLOOD AT REST

(Barometer 760 mm Hg Temp 37 C)

Gas	*Inspired Air*		*Expired Air*		*Alveolar Air*		*Blood*			
							Arterial		*Venous*	
	mm Hg	*%*	*mm Hg*	*%*	*mm Hg*	*%*	*mm Hg*	*Vol %*	*mm Hg*	*Vol %*
O_2	158	20 8	116	15 2	101	13 3	100	19	40	14
CO_2	0 3	0 03	28	3 6	40	5 3	40	52	46	58
N	596	78 4	569	75 0	572	75 2	570		570	
Water Vapor	5	0 7	47	6 2	47	6 2	47		47	

(From Best C H and Taylor N B *The Physiological Basis of Medical Practice* Baltimore Williams & Wilkins 1943)

In the tissues the reverse occurs—blood takes up CO_2 and gives up oxygen

Curves have been plotted to demonstrate these facts The curves show the extent to which hemoglobin is saturated with oxygen at different tensions of O_2 and CO_2 (Figure 6) A decrease of the pH of blood causes a shift and flattening of the curve to the right That is, acidotic blood gives up its oxygen more readily than alkalotic blood An increase in temperature has a similar effect

The amount of oxygen taken up at a given tension of oxygen also varies with the amount of hemoglobin present

Air contains oxygen at a partial pressure of about 158 mm Hg Because of dilution with water vapor, nitrogen and carbon dioxide, alveolar pO_2 is reduced to 100 mm Hg This tension is transmitted without appreciable

decrement to arterial blood A pO_2 of 100 mm Hg sat urates blood at 37 C with a pH of 7 40 to 97 5% One gram of hemoglobin is capable of carrying 1 34 cc of oxygen Thus blood containing 15 gms of hemoglobin per 100 cc will carry 20 cc of oxygen when it is 97 5% saturated The pO_2 of venous blood is approximately 40 mm Hg At this tension the blood is 75% saturated There fore, it has lost 5 cc of O_2 per 100 cc of blood to the tissues

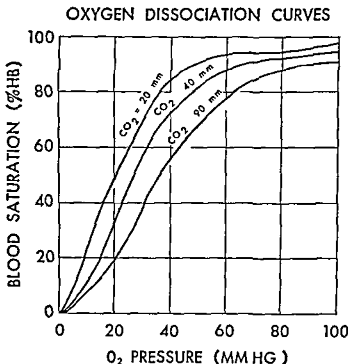


Figure 6

B Dissolved Oxygen Oxygen is also carried in the water of the blood in a dissolved state The amount is proportional to the oxygen tension, 0 3 cc of oxygen is dissolved in 100 cc of blood at 37 C at a pO_2 of 100 mm Hg At a pO_2 of 40 mm, 0 12 cc is carried in the dis

solved state Therefore 0 18 cc of oxygen is given up to the tissues from the blood water Note that this is only 3 5% of the total of 5 cc taken up by the tissues from 100 cc of blood

At a partial pressure of 673 mm Hg (760 mm -87 mm for CO_2 and water vapor) in the alveoli, obtained by breathing 100% oxygen, 2 2 cc more are carried by the blood (0 5 cc as HbO_2 and 1 7 cc in the dissolved state) producing an 11% increase in the total amount of oxygen in arterial blood If the tissues continue to consume 5 cc of oxygen per 100 cc of blood, then the extra 2 2 cc of oxygen will be taken off, plus an additional 2 8 cc and the saturation of the venous blood will be reduced only to 88%, or a pO_2 of 60 mm Hg Therefore, breathing 100% oxygen has increased the amount of oxygen carried in the blood by 11% but has increased by 50% the pressure head of oxygen to which the tissues are exposed

II TRANSPORT OF CO_2

For every mole of oxygen consumed by the tissues 0 82 moles of carbon dioxide are produced Carbon dioxide diffuses from the tissues into the blood primarily as dissolved CO_2 at a rate which is approximately 300 times that of oxygen The venous blood carries 58 cc of CO_2 per 100 cc at a partial pressure of 46 mm Hg This CO_2 is transported by the blood in the following manner

A Only 3 cc of CO_2 per 100 cc of blood can be carried as dissolved CO_2 in the blood at body temperature Therefore 55 cc of the 58 cc in the venous blood must be in chemical combination

B About 10 cc of CO_2 per 100 cc of blood combines directly with the hemoglobin to form carbamino-hemoglobin compounds These compounds are readily formed

decrement to arterial blood A pO_2 of 100 mm Hg saturates blood at 37° C with a pH of 7 40 to 97 5% One gram of hemoglobin is capable of carrying 1 34 cc of oxygen Thus blood containing 15 gms of hemoglobin per 100 cc will carry 20 cc of oxygen when it is 97 5% saturated The pO_2 of venous blood is approximately 40 mm Hg At this tension the blood is 75% saturated Therefore, it has lost 5 cc of O_2 per 100 cc of blood to the tissues

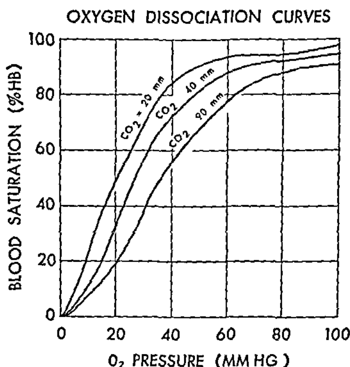


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The oxygen which is carried in arterial blood is osmotically inactive The carbon dioxide carried in venous blood is active osmotically Since it is formed in the red cells and is exchanged ion for ion by chloride, the number of osmotically active elements within the cells of venous blood is higher than that of arterial blood and water must enter the cells This contributes to the production of a higher hematocrit in the venous blood than that in the arterial blood

when oxygen is given up by the hemoglobin in the tissues CO_2 is displaced by oxygenation of the hemoglobin molecule in the lungs

C The remainder is carried as bicarbonate This important reaction occurs in the following manner The CO_2 is dissolved in water to form H_2CO_3 in the red cells ($CO_2 + H_2O \rightleftharpoons H_2CO_3$) This is a slow reaction but the presence of an enzyme or catalyst, carbonic anhydrase in the cells accelerates the reaction about 1000 times The carbonic acid combines with base to form bicarbonate ($H_2CO_3 + B^+ \rightleftharpoons BHCO_3 + H^+$)

Although all of the bicarbonate is formed in the red cells and although hemoglobin buffers most of the hydrogen ion so formed, only 13% of the CO_2 is actually carried in the cells, the greatest part of it is transported in the plasma This occurs because the red cell membrane is permeable only to the anions bicarbonate and chloride As the concentration of bicarbonate increases within the cells bicarbonate begins to diffuse out into the plasma This has the effect of leaving the cells relatively positively charged and chloride diffuses into the cells to preserve electrical neutrality This is called the chloride shift At the lungs the reverse occurs The fact that HbO_2 is a relatively stronger acid than Hb must also be considered For every molecule of HbO_2 reduced in the peripheral circulation 0 7 mM of acid (or H ion) can be absorbed by the weaker acid, Hb This will to a large degree neutralize the acid formed from CO_2 increase At the lungs where HbO_2 is increased the reverse occurs to maintain very limited changes in pH for the CO excreted The Hb therefore also serves a very important function in maintaining the acid base balance of the organism

capnia must exist for cardiac arrest from reflex vagal activation to occur The activity of the cardiac center in the medulla is influenced by afferent impulses from peripheral receptors located at the carotid sinus, aortic arch, great veins, the heart itself and other somatic and visceral receptor organs The centers are also influenced by the respiratory gases, directly or reflexly through carotid and aortic chemocreceptors Important cardiovascular reflexes during anesthesia are discussed in another section

II Chemical Control

A Ions

1) Potassium promotes relaxation of the myocardium When present in excessive amounts it causes progressive heart block first in the auricle then at the A-V node and finally in the ventricle Excessive potassium also produces ventricular arrhythmias and ventricular fibrillation

2) Calcium increases contractility and prolongs the systolic phase Deficiency of calcium decreases the force of cardiac contraction

3) Sodium is essential for excitability and contractility but the manner of its action is not clear

B Epinephrine and Acetylcholine Effects are the same as described in the section on neural control

C Respiratory Gas Tensions in the Arterial Blood

1) Oxygen Oxygen lack accelerates the heart rate and increases the cardiac output through the chemoreceptors and the medullary cardiac centers When the oxygen lack is profound or prolonged the heart rate is slowed from depression of the center and the automatic tissues of the heart Contractility of the myocardium also decreases and cardiac output falls

2) Carbon Dioxide Carbon dioxide excess stimulates the vasmotor center and the cardiac center The heart

VII

HEART — NEURAL AND CHEMICAL CONTROL, CORONARY CIRCULATION

NEURAL AND CHEMICAL CONTROL OF THE HEART

I Neural Control The heart is under the continuous influence of nervous impulses originating in the medulla

A SYMPATHETICS The effect of sympathetic activation is acceleration and increased force of contraction Conduction is also facilitated Sympathetic supply to the heart leaves the spinal cord from the segments T1 to T5 From the cervical sympathetic chain and the upper five thoracic segments fibers join with the cardiac fibers of vagus nerves to form the cardiac plexus The postganglionic fibers supply the automatic tissues and the myocardium itself Transmission is by means of epinephrine like substances

In high spinal anesthesia the heart rate is probably slowed because of blockade of the accelerator fibers and reduced adrenal secretion

B PARASYMPATHETICS The cardiac fibers of the right vagus end in the S A node and those of the left in the A-V node Vagal activation slows the sinus rhythm and the A-V conduction Transmission is mediated by acetylcholine secreted at the myoneural junction Reflex activation of the vagus nerve slows the heart and is thought by some to be responsible for cardiac arrest during anesthesia However, it is probable that anoxia and hyper-

The heart is therefore rendered capable of caring for an increased venous return Other effects of digitalis on the heart are the increase of mechanical efficiency of the muscle so that a given amount of work can be done with less consumption of oxygen, the slowing of the heart rate by vagal stimulation and the slowing of A-V conduction

2 Quinidine is a depressant drug on heart muscle It decreases myocardial irritability and increases the refractory period of the heart muscle with the result that the heart rate slows

CORONARY CIRCULATION

An intact coronary circulation is essential for the normal function of the heart Coronary insufficiency occurs when 1) the blood flow is reduced because of lowered perfusing pressure of the coronary arteries, or 2) the work load of the heart is increased out of proportion to the available blood supply Normal coronary circulation is determined by many factors

1 *Coronary Arterial Blood Pressure* The flow increases as the pressure is increased As the coronary arterial pressure falls the flow changes in the same direction Below a critical level, 10 to 15 mm Hg the inflow ceases

2 *Extravascular Support from Myocardial Tonus and Contraction* The coronary flow is highest during diastole and lowest during systole

3 *Neural Influences* The vagus nerves contain constrictor fibers and sympathetic nerves contain dilator fibers However it is difficult to establish conclusively the separate effect of nervous influences upon the coronary vessels

4 *Local Oxygen Tension and Concentration of Metabolites* Lowered oxygen tension is the most potent

rate is usually not greatly increased Conductivity is also depressed Carbon dioxide has a direct action on the myocardium The extensibility of the myocardium during diastole is enhanced, favoring the filling of the heart The stroke output is therefore increased However, continued exposure to high CO_2 tension reduces contractility and also predisposes to arrhythmias

3)pH Decrease of the pH causes slowing of the rate, depresses the conductivity and enhances relaxation of the myocardium Mammalian heart *in situ* stops in diastole when the blood pH is decreased to 6 25

III *Drugs*

A Anesthetic Agents

Ether, chloroform, cyclopropane, tribromethanol and thiopental in excessive concentrations depress the conductivity and contractility of the heart Overdosage of these anesthetic agents is a common cause of cardiac arrest and circulatory collapse The local anesthetic agents cocaine procaine and others also depress the myocardium Their action on the heart is similar to that of quinidine Excessive concentration of local drugs in the blood causes circulatory collapse from myocardial depression and peripheral vasodilatation Cyclopropane, chloroform and ethyl chloride increase the irritability of the myocardium They favor the occurrence of ventricular arrhythmias in the presence of epinephrine, endogenous or administered (Other vasopressor amines with a dihydroxybenzene nucleus the catechol nucleus, i e, norepinephrine and isopropyl norepinephrine, have effects similar to those of epinephrine)

B Other Drugs

1 Digitalis acts on the myocardium to increase the force of systolic contraction and ventricular emptying

congestive heart failure occurs The cardiac output may be above or below the normal value

Normally the heart compensates for increased venous return or peripheral resistance by diastolic lengthening of the myocardium—dilatation The increased diastolic volume of the heart will increase the stroke volume and thus accommodate the increased venous return or overcome the increased peripheral resistance If these conditions persist, myocardial hypertrophy will occur Hypertrophy is caused by an increase in the size of individual fibers but not the number of fibers The available blood supply poses a limit to the degree of hypertrophy, beyond which relative coronary insufficiency will occur

When the function of the myocardium is impaired by coronary insufficiency, disease of the myocardium or drug depression, or when sudden increase of the work load is imposed on a heart with no reserve for compensation, the diastolic volume will increase even further A point will be reached when the contractions are no longer efficient because of overstretching of the myocardial fibers and failure will occur Therapy is directed toward the improvement of performance of the myocardium with digitalis and the reduction of the work load of the heart by simulated or actual venesection

ARRHYTHMIAS

The most common arrhythmias during anesthesia are

1 Extrasystoles of various origin
2 Displaced pacemaker

Arrhythmias can occur with any general anesthetic agent but they are most common during deep cyclopropane anesthesia Most of the time the arrhythmias are fleeting and subside without any specific treatment Ven-

coronary dilator The increase in coronary flow precedes changes in heart rate or blood pressure during anoxia Maximal dilatation occurs when the arterial oxygen saturation falls to 50% (for heart *in situ*) Increased concentration of metabolites locally also causes dilatation Normally, the coronary flow is automatically adjusted to *the requirement of the myocardium* Inhalation of 5 to 8% carbon dioxide has no significant effect on coronary flow

EFFECT OF DRUGS ON THE CORONARY CIRCULATION

Papavarine xanthines, nitrites and systemic vasopressors are vasodilators for the coronary vessels However the change in coronary flow is always associated with concomitant changes in the force of contraction, metabolism and work of the heart It is difficult to draw the conclusion that the dilatation is solely attributable to the action of drugs on the vessels Increased metabolism resulting from increased work can be partly or wholly responsible for the change in coronary flow

Pitressin has a powerful constrictor effect on coronary vessels If there is peripheral hypertension and an increased workload imposed on the heart, coronary constriction with reduced blood flow is extremely dangerous Fatalities during anesthesia have been reported from administration of pitressin or posterior pituitary extract in obstetrical practice These drugs should never be used *during any general anesthesia* Pitocin *the oxytocic factor* of the posterior pituitary gland, does not have any vascular action and is safe to use

HEART FAILURE

When heart action is not efficient enough to maintain an adequate circulation for the requirements of the body,

VIII

PERIPHERAL CIRCULATION

HEMODYNAMIC CHANGES AND IMPORTANT CARDIOVASCULAR REFLEXES DURING ANESTHESIA

THE INTEGRITY of the peripheral circulation depends on

1 Adequate circulating blood volume
2 Intact function of the heart
3 Intact regulatory mechanisms for the maintenance of homeostasis throughout the body

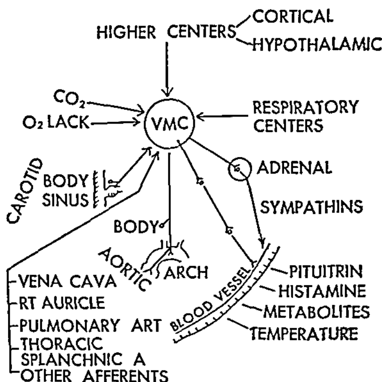
All of these factors can change greatly during anesthesia and surgical procedures Depth of anesthesia, hemorrhage and reflex vascular responses to surgical manipulation all have profound effects on hemodynamics The blood volume can be measured with relative accuracy with any of the current techniques, i e dilution methods with dyes or labeled red blood cells However, in the evaluation of the patient s circulatory status, the blood volume is only one of the factors and can not be relied upon alone Measurements of the arterial pressure and pulse rate are still clinically valuable guides of the integrity of the peripheral circulation

Factors affecting the blood pressure (other factors remaining the same)

tricular arrhythmias are potentially dangerous since they may lead to ventricular fibrillation if the condition is not corrected Hypoventilation may play a major role in the productivity of arrhythmias Inadequate ventilation should always be corrected Intravenous injection of pro caine, procaine amide or quinidine may terminate some types of ventricular arrhythmias In cyclopropane ar rhythmias, lightening of anesthesia and addition of ether to the anesthetic mixture will usually restore normal rhythm

Tachycardias with ventricular rates faster than 160 per minute may reduce the efficiency of the heart and decrease the cardiac output because of shortened diastole and inadequate filling Sinus tachycardia can be slowed by ocular or carotid sinus pressure, but it returns when the maneuver is discontinued Paroxysmal auricular tachycardia can often be converted to sinus rhythm by these measures In ventricular tachycardia such measures can only slow the rate temporarily but cannot convert the rhythm If this arrhythmia persists procaine amide, procaine or quinidine are given

Ventricular fibrillation is a rare complication This condition as well as cardiac arrest has to be treated *promptly* *Three minutes* is the limit for the central cortical structures to survive without damage In cases where hypoxia is already present before the catastrophe, the time limit will be shorter The procedures for cardiac re suscitation are discussed in another section



REGULATION OF ARTERIAL TONE

Figure 7 (From Wright, S *Applied Physiology* 9th Edition Oxford University Press 1952)

through the vagus nerve The carotid baroreceptors, with those in the aortic arch play an important role in the regulation of arterial pressure An elevation of intrasinual pressure stimulates these baroreceptors Afferent impulses are inhibitory in nature, cause reflex bradycardia and vasodilatation and inhibition of respiration A fall in the intrasinual pressure reduces the amount of these inhibitory afferent impulses and results in tachycardia and vasoconstriction

3 OTHER BARORECEPTORS FROM VENAE CAVAE AND THE RIGHT AURICLE

	Systolic Pressure	*Diastolic Pressure*	*Pulse Pressure*
Increased peripheral resistance	+	++	−
Decreased peripheral resistance	−	−−	+
Increased heart rate	+	++	−
Decreased heart rate	−	−−	+
Increased systolic discharge	++	+	+
Decreased systolic discharge	−−	−	−
Decreased arterial distensibility	+	−	+
Increased arterial distensibility	−	+	−
Increased blood volume	++	+	+
Decreased blood volume	−−	−	−

(+ indicates an increase — indicates a decrease)

REGULATION OF ARTERIAL TONE

The arterial tone is constantly under the influence of neural impulses, circulating hormones and local metabolites The schematic diagram, as modified from Wright's *Applied Physiology,* indicates different factors controlling the arterial tone (Figure 7)

I Neural Impulses

A The vasomotor centers are located in the caudal part of the medulla Factors affecting activities of the vasomotor centers are

1 Higher Centers

a Cortical impulses Fear, anxiety and emotional disturbance influence the arterial pressure and heart rate through the vasomotor centers

b Hypothalamic autonomic center and heat regulating centers

2 Afferent Impulses from Carotid and Aortic Arch Baroreceptors The carotid sinus nerves ascend to the medulla through the glossopharyngeal nerve, the aortic arch baroreceptors send their impulses centrally

diastolic pressure, so the pulse pressure is increased The pulse rate is not changed or only slightly increased because of the baroreceptor reflexes and action of carbon dioxide on the S-A node The local effect of CO_2 excess on the vessels is vasodilation but this effect is matched by the greater central effect

b Oxygen lack stimulates the center reflexly through the chemoreceptors The direct effect of hypoxia on the center is that of depression Oxygen excess has no apparent effect on the activity of the vasomotor center

5 ACTIVITIES OF THE RESPIRATORY CENTER SPREAD OVER TO THE VASOMOTOR CENTERS

B Efferent pathways from vasomotor centers to the peripheral vessels descend to the neurones in the ventro lateral column of the dorsal and lumbar spinal cord Motor neurones in the ventro lateral column have fibers which emerge from the spinal cord with the ventral roots, leave the ventral roots through the white rami communicans Post-ganglionic fibers leave the paravertebral sympathetic ganglia to supply the viscera and blood vessels The preganglionic fibers also supply the adrenal medulla Recent experiments indicate that the sympathetic adrenergic constrictors are the sole pathway for the control of peripheral vascular resistance In the dog sympathetic adrenergic dilators, sympathetic cholinergic dilators, and dorsal root dilators cannot be demonstrated to have any physiological significance[4]

II Hormonal Influence

A *Adrenal Medullary Hormones* Epinephrine and norepinephrine secreted by the adrenal medulla have profound effects on the circulation

Epinephrine in physiological concentrations or when given in small doses in continuous intravenous infusion

Bainbridge Reflex[1] An increase in heart rate is induced by a rise in the pressure of the blood entering the right auricle due to stimulation of nerve endings in the walls of the great veins and under the endocardium of the right auricle

McDowall Reflex[2] A fall in the venous pressure stimulates nerve endings in the right auricle which send impulses to the medullary centers with resulting generalized vasoconstriction This is partially responsible for the vasoconstriction which occurs in hemorrhage and shock

It has also been shown by McDowall that a rise in venous pressure considerably above normal causes a vago pressor reflex with vascostriction This reflex occurs coincidentally with and is supplemental to Bainbridge It antagonizes the depressor reflexes and, through predominance over them, the elevation of the arterial pressure is permitted to continue throughout exercise

Harrison Reflex[3] An increase in venous pressure in the right auricle causes an increase in respiratory rate This same effect is not obtained with the vagi cut

Pulmonary arteries, thoracosplanchnic arteries, and other afferents (somatic and visceral stimulations) also influence the vasomotor centers

4 Changes in Arterial pH, Carbon Dioxide Tension and Oxygen Tension

a Carbon dioxide excess and increased H ion concentration stimulate the vasomotor center mostly through the chemoreceptors The center is stimulated directly only by a very high concentration of carbon dioxide The arterial pressure is markedly increased because of peripheral vasoconstriction and increased stroke volume of the heart (probably from direct action of carbon dioxide on the heart) The systolic pressure is raised more than the

mains relatively unchanged The cardiac output tends to fall if there is no excitement during the induction period The total peripheral resistance is increased The resistance of the renal and the splanchnic vascular bed increases markedly and the blood flow through the kidney and other splanchnic organs is decreased[5 6] Measurements of blood flow of the skin and muscles indicate that there is marked vasodilatation in these vascular beds Cerebral circulation and coronary circulation are not significantly altered

In deep or prolonged anesthesia, the cardiac output is further reduced with a decrease in the total peripheral resistance and a fall in arterial pressure

The mechanism of these profound hemodynamic changes is not clearly understood although the available evidence indicates that some of these changes are probably humoral in origin

Inexpertly administered anesthesia can cause circulatory complications other than the normal changes just described Overdosage of anesthetic agents depresses cardiac function and vasomotor tone to a greater extent Patients with poor myocardial function or deficient blood volume are more susceptible to the depressant action of anesthetic agents Hypoventilation has a deleterious influence on the circulation Finally, hemorrhage, trauma and cardiovascular reflexes from surgical manipulations may initiate additional disturbances All these factors should be considered and correlated in order to arrive at a correct diagnosis and to determine the appropriate management of any change in circulation during anesthesia

B Shock Arterial pressure is maintained by reflex vasoconstriction in the earliest stages of shock from hemorrhage or trauma The tissues and the vessels suffer anoxia as a result of the reduced and sluggish blood flow during vasoconstriction If the condition is not treated, secondary dilatation occurs from anoxic damage Blood

or hypodermic injection, causes overall vasodilatation However, it also increases the heart rate and stroke volume resulting in increased cardiac output, reduced total peripheral resistance, increased systolic pressure and lowered diastolic pressure

Norepinephrine on the other hand, is a potent vasoconstrictor The total peripheral resistance is increased, the diastolic pressure increased more than the systolic pressure The cardiac output is reduced and the heart rate is slowed reflexly by the hypertension

B Thyroxin sensitizes the blood vessels to the effect of epinephrine In cases of thyrotoxicosis, the release of thyroxin upon manipulation of the thyroid gland can result in marked hypertension and tachycardia This complication will sometimes respond to one mgm doses of Hydergin® (di hydroergocristine, cornine,-kryptine) a sympatholytic drug, or Gynergen® (ergotamine tartrate) 1 to 3 mgm intravenously

C Pitressin is a potent vasoconstrictor Its use is dangerous because of its constrictor effect on the coronary vessels

Local anoxia, carbon dioxide excess, change in H ion concentration, as well as accumulation of metabolites, serve to dilate vessels locally

III Hemodynamic Changes and Common Circulatory Disturbances During Anesthesia

A Hemodynamic Changes During General Anesthesia During general anesthesia, certain aspects of cardiac function and peripheral hemodynamics undergo marked derangements These changes seem to be associated with the state of anesthetization since they occur during well conducted anesthesia and there are inconsequential differences among the various anesthetic agents

In light surgical anesthesia, the blood pressure re-

afferent nerves by manipulation or traction of abdominal viscera may give rise to the celiac plexus reflex syndrome There are circulatory and respiratory components The circulatory component is manifested by a drop in arterial pressure, predominantly the systolic pressure, with a very narrow pulse pressure The pulse rate usually is slowed Apnea followed by hyperpnea supervenes There is also abdominal muscular rigidity The usual treatment is the intravenous administration of ephedrine Deepening of the anesthesia or interruption of the afferent pathway by celiac plexus block can also correct this condition

5 Reflexes from Somatic Nerve Stimulation Manipulation of major somatic nerve trunks or subperiostial dissections can cause either a rise or fall of the arterial pressure, depending upon the intensity and the type of the stimulus, the depth of anesthesia and the existing cardiovascular status at the time of stimulation

REFERENCES

1 Bainbridge F A On some cardiac reflexes *J Physiol* *48* 332 340 1914

2 McDowall R J S A vago pressor reflex *J Physiol*, 59 41 47 1924

3 Harrison T R Harrison W G and Marsh J P Reflex stimulation of respiration from increase in venous pressure *Am J Physiol* *100* 417 419, 1932

4 Frumin, M J, Ngai, S H and Wang S C Mechanism of vasodilatation in the hind limb of dogs *Federation Proc* *11* 51 52, 1952

5 Habif, D V, *et al* Renal and hepatic blood flow glomerular filtration rate and urinary output of electrolytes during cyclopropane ether and thiopental anesthesia operation and immediate postoperative period *Surgery*, 30 241 255 1951

6 Papper E M Renal function during general anesthesia and operation *J A M A*, *152* 1686-1689, 1953

is pooled in the capillaries and removed from the active circulation Plasma fluid is lost to the extravascular space A vicious cycle is started and shock reaches the irreversible stage

Use of vasopressor drugs in the treatment of shock is not a rational therapy except as an emergency measure when blood, plasma, or plasma expanders are not immediately available

C Important Cardiovascular Reflexes During Anesthesia

1 OCULO VAGAL REFLEXES Excessive pressure on the eyeball or manipulation of the eyeball can result in vagal slowing of the heart and hypotension Pressure on the supratrochlear nerve has a similar effect

2 CAROTID SINUS REFLEX Inadvertent pressure or manipulation of the carotid sinus stimulates the baroreceptors and can result in marked bradycardia and profound hypotension Belladonna drugs in adequate dose (1 5 2 0 mgms) effectively block the vagal effect on the heart but do not alter the vascular response This reflex is often used in the treatment of paroxysmal tachycardia of supraventricular origin by digital pressure on the carotid sinus region

3 VAGO VAGAL REFLEXES Stimulation of afferent vagal or glossopharyngeal nerve endings during the manipulations of laryngoscopy endotracheal intubation, endotracheal suctioning bronchoscopy, and visceral traction can result in reflex slowing or stoppage of the heart It is probable that this reflex becomes more dangerous if there is hypoxia or hypercapnea Recent experimental evidence indicates that hypoxia and hypercapnea sensitize the myocardium to reflex vagal stimulation Serious ventricular arrhythmia and even cardiac asystole may ensue

4 CELIAC PLEXUS REFLEX Stimulation of visceral

mechanisms achieve the rapid and diffuse reactions. 1) Anatomically, each preganglionic sympathetic fiber comes into synaptic relationship with many postganglionic fibers and thus activates a large area. 2) Some preganglionic fibers supply the adrenal medulla. Activation of the adrenal medulla causes outpouring of epinephrine and norepinephrine (the sympathetic mediator) which are circulated throughout the body. In man the sympathetic outflow arises from the spinal cord from the thoraco-lumbar region, extending from T1 to L2.

B. The parasympathetic system is generally concerned with the protection, restoration and conservation of the bodily resources. The preganglionic fibers end close to or within the organ supplied. The responses are more restricted in localization. The parasympathetic (cranio-sacral) outflow has its origin from the hypothalamus, midbrain, pons, medulla, and sacral spinal cord.

For detailed anatomical and physiological considerations textbooks and monographs on the subject should be consulted.

CHEMICAL MEDIATION OF AUTONOMIC NERVOUS IMPULSES AND PHARMACOLOGY OF THE AUTONOMIC NERVOUS SYSTEM

When impulses reach the nerve endings in the synaptic junction in the autonomic ganglia or neuro-effector junction humoral substances are liberated. The liberated chemical substances serve to activate the ganglionic neurones and thus transmit the impulses to postganglionic fibers, or in the case of the neuro effector junction, the chemical substance activates the effector organ. The available evidence indicates that the ganglionic and postganglionic parasympathetic transmission is mediated by *acetylcholine*, and in the case of postganglionic sympa-

IX

AUTONOMIC NERVOUS SYSTEM

THE AUTONOMIC nervous system is that part of the nervous system innervating smooth muscles, cardiac muscle and glands It is primarily concerned with the adjustments of the internal environment of the body It has neural centers in the central nervous system at various levels and peripherally, pathways to and from the effector organs Its activity is for the most part not under voluntary control and it operates below the level of consciousness However it will be erroneous to assume that the activity of the autonomic nervous system is independent of somatic function Somatic and visceral function are interdependent and cortical activity influences both systems

The peripheral autonomic nervous system consists of efferent and afferent fibers The afferent fibers are concerned with visceral pain and autonomic reflexes The efferent autonomic pathways differ from the somatic pathways in that they have synapses outside of the central nervous system *Preganglionic fibers* end among neurones in the *ganglia* and from these ganglia impulses are conducted to the effector organs through *postganglionic fibers*

The efferent system can be divided into two components the *sympathetic* and the *parasympathetic system*

A The sympathetic system is generally activated when the body is under stress The resulting adjustment is rapid and widespread involving many organs Two

usually administered as a continuous intravenous infusion (0 1 0 2%) The blood pressure usually recovers within 30 minutes after the infusion is discontinued

5 D TUBOCURARINE has ganglionic blocking action in large dosage

6 SUCCINYLCHOLINE Two to three hundred times the paralyzing dose of succinylcholine stimulates ganglionic neurones (or facilitates ganglionic transmission) and results in hypertension[6] Seven hundred times the paralyzing dose blocks ganglionic function and results in hypotension Clinically hypertension is seen during the administration of this drug in some 20% of the cases

7 ACETYLCHOLINE Although in physiological concentration it serves as a mediator, in high concentration it blocks ganglionic transmission because of the persistent depolarization of ganglionic neurones

8 DI ISOPROPYL-FLUORO PHOSPHATE (DFP), TETRAETHYL PYRO PHOSPHATE (TEPP) AND OCTA-METHYL-PYROPHOSPHOR-AMIDE (OMPA) These agents are anti cholinesterases In low doses they facilitate and in high doses they block ganglionic transmission

B Sympathetic Mediators At the nerve endings of the postganglionic sympathetic fibers the substances liberated have properties and activity similar to that of epinephrine and norepinephrine The response of the effector organs can be either excitatory (sympathin E, or E receptors) or inhibitory (sympathin I, or I receptors) The destruction of the sympathetic mediators takes place at a limited rate and consequently they may diffuse to other structures or into the blood stream

1 SYMPATHOMIMETIC AGENTS

a Norepinephrine—excitatory
b Epinephrine—inhibitory and excitatory

thetic transmission, by epinephrine like substances[1 2 3] Many drugs have activity similar to these mediators (mi metic) and many others block their action on the effector organ (-lytic) In addition, those drugs which influence the activity of an enzyme system assisting the removal of the mediator, also have profound effects on the transmis sion of neural impulses For instance, acetylcholine is hydrolyzed rapidly by the enzyme, cholinesterase Anti cholinesterase drugs slow down the rate of hydrolysis of acetylcholine and have activity mimetic to acetylcholine The discussion can be divided into three categories

A Ganglionic Transmission Any drug which influ ences the ganglionic transmission modifies the activity of both the sympathetic and parasympathetic systems

1 Nicotine A ganglionic blocking agent The block-ade is preceded by a preliminary phase of stimulation

2 Tetraethylammonium Chloride (TEA) blocks the ganglionic transmission but its effect is transient and incomplete

3 Hexamethonium (C_6) and Pentamethonium (C_5) block ganglionic transmission[4] They are used in anesthesia for the induction of hypotension under certain circumstances In addition to hypotension these drugs also cause moderate tachycardia flushed and dry skin and loss of autonomic reflexes The last is of particular import ance because without reflex regulation of circulation these patients cannot tolerate the erect position

4 Arfonad The action of Arfonad is largely due to ganglionic blockade although there is evidence to indicate that it also has a direct vasodilator action[5] It is used for the induction of hypotension during anesthesia Its dura tion of action is shorter than that of C_5 and C_6 and is

2 Sympatholytic Agents (Adrenergic Blocking Agents) These agents block the response of the effector organs to the excitation of sympathetic mediators There is no compound which blocks the inhibitory effect The sympathetic agents are useful in the treatment of thyroid storm, the management of pheochromocytomas and for the prophylaxis and treatment of cardiac arrythmias

a Dibenamine—its action is potent and prolonged

b Ergot alkaloids (Gynergen® [ergotamine tartrate], Hydergin® [di hydroergocristine, -cornine-kryptine])

c Benzodioxanes The blockade produced by these compounds is rather weak and readily overcome by large doses of epinephrine The duration of action is relatively short

d Regitine® (Phentolamine) is important in the diagnosis and therapy of pheochromocytoma Use during surgery will prevent paroxysms of hypertension when the tumor is manipulated

e Priscol (Imidazoline derivative) The duration of action is very short

C Parasympathetic Mediators Postganglionic parasympathetic impulses are mediated by acetylcholine Although ganglionic transmission and neuromuscular transmission are mediated by the same substance, drugs affecting the acetyl choline cholinesterase system do not have the same activity at these different sites For instance d Tubocurarine blocks neuromuscular transmission and with higher doses, blocks ganglionic transmission, but it does not influence postganglionic cholinergic transmission Atropine blocks postganglionic cholinergic activity and hexamethonium blocks ganglionic transmission but they do not block neuromuscular transmission

1 Parasympathomimetic (Cholinergic) Agents

c Iso propyl norepinephrine (Isopryl)—inhibitory
d Neosynephrine—excitatory
e Ephedrine There is evidence that ephedrine does not have a direct action on the effector organ but acts through competitive inhibition of amine oxidase,[7] the enzyme responsible for the destruction of sympathetic mediators
f Other vasopressor amines

The over all cardiovascular effect of these agents depends on the relative preponderance of their action on the heart and blood vessels and on cardiovascular reflex activity For example, Neosynephrine causes profound peripheral vasoconstriction but has very little action on the heart The heart rate is reduced because of baroreceptor reflexes Epinephrine affects the heart as well as the peripheral vessels The heart rate and cardiac output are increased However, the blood pressure response is a function of the dose With small doses, there is vasodilatation and decreased diastolic pressure and little increase in systolic pressure With larger doses both the diastolic and systolic pressure are increased because of peripheral vasoconstriction and more marked increase of the cardiac output Iso propyl norepinephrine lowers the blood pressure due to vasodilatation but it causes tachycardia because of its direct and reflex actions on the heart

Sympathomimetic agents with inhibitory activity also relax smooth muscles of the bronchioles intestines and the uterus The drugs with a *catechol* nucleus (di hydroxybenzene nucleus, i e, epinephrine norepinephrine and iso propyl norepinephrine) increase the irritability of the myocardium and are contraindicated during cyclopropane, chloroform, and trichlorethylene anesthesia

Site 5—d-Tubocurarine, decamethonium (Syncurine), succinylcholine

Site 7—and Site 4 (sweat glands) atropine, scopolamine

REFERENCES

1 Loewi O Uber Humorale Übertragbarkeit der Hertz nervenwirkung *Arch ges Physiol*, *189* 239 242 1921

2 Feldberg, W and Vartiainen, A Further observations on physiology and pharmacology of sympathetic ganglion *J Physiol*, *83* 103 128, 1934

3 Cannon, W B and Lissak K Evidence for adrenalin in adrenergic neurones *Am J Physiol* *125* 765 777 1939

4 Paton W D M and Zaimis E J Pharmacological actions of polymethylene bistri methylammonium salts *Brit J Pharmacol*, *4* 381 400, 1949

5 McCubbin J W and Page I H Nature of hypotensive action of thiophonium derivative (Arfonad RO2 2222) *Anaesthesia* *8* 175-181, 1953

6 Thesleff, S Pharmacological properties of succinylcholine iodide with particular reference to its clinical use as muscular relaxant *Acta physiol scandinav* *26* 103-129 1952

7 Gadum J H and Kwiatkowski H The action of ephedrine *J Physiol*, *94* 87 100 1938

a Acetylcholine and its derivatives

b Anticholinesterases — physostigmine, prostigmine, DFP, TEPP and OMPA Mytolon® and Tensilon® also have anticholinesterase activity and act chiefly on neuro muscular transmission

2 PARASYMPATHOLYTIC (CHOLINERIC BLOCKING AGENTS Belladonna derivatives, atropine, scopolamine and related synthetic compounds In anesthesia these agents are used to block salivary and mucous gland secretion A larger dose is required to block the cardiac vagus than to produce drying of secretions

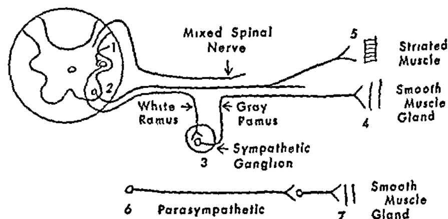


Figure 8

Acetylcholine Mediator at all sites 1, 2, 3 5, 6, 7 and 4 if this is a sweat gland

Norepinephrine or Epinephrine like Substances Mediator at site 4 except sweat glands

Blocking Compounds

Site 3—tetraethyl ammonium ion hexamethonium, pentomethonium, d Tubocurarine

Site 4—(except sweat glands) ergot derivatives (Hydergin) Dibenamine, benzodioxane etc

noid tissue is on the posterior wall The mucous membrane of the respiratory portion of the nose is ciliated epithelium and is thick over the inferior turbinates where it is very vascular and can act as erectile tissue, swelling sufficiently during infections or allergic states to block the airway completely The posterior portion of the inferior turbinate may also be the site of polyps blocking the choanae

II MOUTH AND PHARYNX

The lips can be traumatized against airways or laryngoscopes The cheeks sink in when teeth are absent and mask fit becomes difficult

The temperomandibular joint is easily dislocated forward by pressure on the chin when the mouth is open The upper teeth are more frequently damaged by instrumentation than the lower teeth

The main substance of the tongue is composed of four intrinsic muscles on each side the superior and inferior longitudinal muscles and the transverse and vertical muscles The tongue is attached to the mandible and hyoid bone by the extrinsic muscles, which relax during anesthesia, allowing the tongue to fall back and lie against the posterior wall of the pharynx The extrinsic muscles of the tongue are

1 *Genioglossus* A flat, triangular muscle the medial surface of which is in contact with its fellow It arises from the upper genial tubercle on the posterior surface of the body of the mandible and spreads out in a fanlike manner to be inserted into the whole length of the tongue Its action is to project the tip of the tongue forward as well as depress the whole organ

2 *Hyoglossus* A quadrate, flat muscle which arises from the whole length of the greater cornu of the hyoid bone and also from its body Its fibers pass upwards to

X

ANATOMY OF THE UPPER RESPIRATORY TRACT, AS RELATED TO PROBLEMS OF AIRWAY IN ANESTHESIA

I NOSE

At the apex of the nose the skin is thick and adherent It is thin over the bridge of the nose and easily injured by undue mask pressure The openings of the nares lie below the level of the floor of the nose, to gain access to the nasal fossae the tip of the nose must be elevated

The septum separating the two nasal fossae consists of the perpendicular plate of the ethmoid, the vomer and the septal cartilages covered by thin, easily traumatized mucous membrane Deviation of the septum is the rule in adults and may occlude one, and rarely both nostrils On the lateral walls of each fossa are the three turbinates, projecting like horizontal shelves and dividing the fossa into meatuses The inferior turbinate lies along the functional airway which is the inferior meatus It becomes swollen in maxillary sinusitis and is easily traumatized by nasal tubes passed along the floor of the nose The floor of the nose is supported by the palatal processes of the maxillary bones and the horizontal plates of the palatal bones In the supine position the direction of the floor of the nose and the inferior meatus is vertical, and it is in this direction that a nasal tube must be inserted

Each nasal fossa opens behind into the nasopharynx posterior to the soft palate The openings of the Eustachian tubes are on the lateral walls and pharyngeal ade-

They converge to form a slender slip that descends in the palatoglossal arch to the back part of the side of the tongue They draw the tongue and soft palate closer together [1]

The posterior, pharyngeal surface of the tongue has lymphoid tissue, the lingual tonsil, which can be the source of bleeding when traumatized Occasionally the tongue is so enlarged as to cause respiratory obstruction by its size alone This can be predicted by the presence of indentations along the edges caused by pressure against the teeth

On the lateral walls of the oropharynx, the tonsils, especially when enlarged may be traumatized or may be large enough to cause respiratory obstruction

Below the oropharynx and behind the dorsum of the tongue is the laryngopharynx, with the epiglottis anteriorly, the elevated entrance to the larynx centrally the esophagus posteriorly, and the pyriform fossae laterally

III LARYNX

The larynx contains three paired cartilages and three unpaired nine in all

Unpaired *The cricoid cartilage*, the only complete ring is the support of the larynx It is narrow anteriorly and broad posteriorly

The thyroid cartilage the Adam s Apple, is superior to the cricoid and attached inferiorly to the cricoid by the cricothyroid ligament and superiorly to the hyoid bone by the thyrohyoid membrane

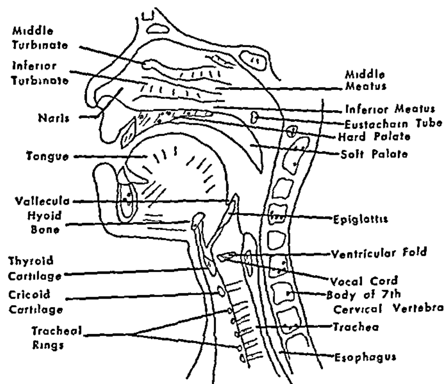
The epiglottis is attached to the thyroid cartilage just above the vocal cords and to the base of the tongue Between its free upper portion and the tongue is a space known as the vallecula

Paired *Arytenoid cartilages* are attached above the

be inserted into the posterior one half of the side of the tongue It helps to depress the tongue and enlarge the cavity of the mouth

3 Styloglossus An elongated slip which takes origin from the styloid process It passes downwards and forwards to be inserted into the whole length of the side of the tongue It pulls the tongue backwards and upwards during swallowing

4 Palatoglossus Forms a thin sheet in the lower part of the soft palate attached to the palatine aponeurosis



SAGITTAL SECTION

(FACE AND NECK)

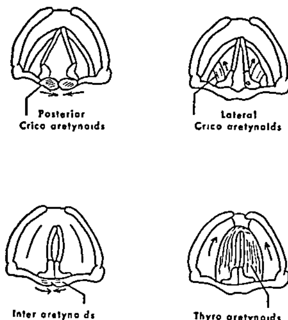
Figure 9

Muscles of the Larynx

The muscles of the larynx are divided into extrinsic and intrinsic The extrinsic move the larynx as a whole, the intrinsic are involved in vocal cord motion including the production of laryngospasm

These muscles may be separated into those which open the vocal cords (abductors) and those which close the cords (adductors) (Figure 11)

Abductors—posterior cricoarytenoid from back of cricoid cartilage to muscular process of arytenoid *abducts*



ACTION OF INTRINSIC
MUSCLES OF LARYNX

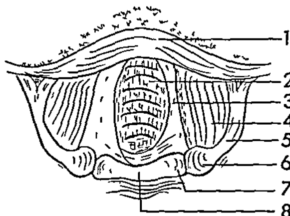
Figure 11 (From Pressman J J Physiology of Vocal Cords in Phonation and Respiration *Archives of Otolaryngology* Vol 35, No 3, 1942)

cricoid close to the midline posteriorly Their anterior angles are the attachments for the vocal cords

Corniculate cartilages lie above the arytenoids, with the *cuneiform cartilages* lateral to them

The vocal cords extend from the thyroid cartilage back to the arytenoids Above them are the ventricles of the larynx, and above these and a little lateral are the ventricular bands

The trachea extends down from the cricoid cartilage as a tube composed of incomplete cartilagenous rings joined by a membrane posteriorly



1 Epiglottis
2 Glottis
3 Vocal Cord
4 False Vocal Cord
5 Ary epiglottic Fold
6 Cuneiform Cartilage
7 Corniculate Cartilage
8 Interaretynoid Fold

LARYNGOSCOPIC VIEW OF LARYNX

Figure 10 (From Thomas G Technique of Intubation Anesthesia with Detailed Illustrations *Current Researches in Anesth & Analg* Vol 17, 1938)

around the latter to the larynx It lies farther from the trachea than does the left recurrent

The left recurrent is given off at the level of the arch of the aorta, around which it curves to the larynx It lies closer to the trachea and esophagus

Behavior of a paralyzed vocal cord depends on what is cut the whole nerve, or a branch supplying one function Clinically, paralyzed cords show one of three patterns

1 Immediate adduction (midline position) when only the supply to the abductors is cut

2 Immediate abduction (extreme opening) when only the supply to the adductors is cut

3 Intermediate position (halfway between adduction and abduction), so called Cadaveric Position seen in 90% of all cord paralysis when an entire recurrent laryngeal is cut This position is seen only in new cases, because the flaccid paralysis of peripheral nerve lesions is followed by atrophy, fibrosis and contraction and over a period of time the action of the unparalyzed cricothyroid muscle gradually pulls the cord toward the midline (unless the superior laryngeal is also cut)

A paralyzed cord which immediately assumes the midline position never shifts out nor does a cord paralyzed in the extreme lateral position ever shift toward the midline

During normal respiration and in light planes of general anesthesia, the vocal cords adduct during inspiration and abduct during exhalation During deep general anesthesia or under the influence of muscle relaxant drugs the reverse occurs

REFERENCE

1 Brash J C and Jamieson E B *Cunningham s Manual of Practical Anatomy* New York, Oxford, 1940

cords by pulling muscular process of arytenoid down and medially, thus raising and everting the vocal process

Adductors—lateral cricoarytenoid from upper lateral cricoid to muscular process of arytenoid *adducts* cords by pulling muscular process forward

Interarytenoid from muscular process of each arytenoid across midline to summit of the other pulls arytenoids together, part of constrictor mechanism

Thyroarytenoid from angle of thyroid back to arytenoid, medial margin lies in vocal cord part of constrictor mechanism but in part relaxes cords

Cricothyroid anterior arch of cricoid to inferior margin of thyroid cartilage elongates and tenses vocal cords

Aryepiglottic few, weak fibers in aryepiglottic folds helps close larynx during swallowing

Note The arytenoids rotate about axes which can move towards and away from the midline producing changes in position and shape of the vocal cords

Nerve Supply of the Larynx

The entire nerve supply to the larynx is from two branches of the vagus

A The superior laryngeal nerve is given off the vagus at the ganglion nodosum It divides into 1) the internal branch which is purely sensory and 2) the external branch which is purely motor and supplies the cricothyroid muscle

B The recurrent laryngeal nerves on the two sides leave the descending vagus trunks at different levels and have different courses but the ultimate distribution is the same

The right recurrent is given off where the descending vagus crosses the right subclavian artery, and curves

Guy Lussac s Law The pressure of a gas is directly proportional to its absolute temperature, when the volume remains constant For each degree of temperature change, the pressure will change 1/273

Avogadro's Law Equal volumes of gases at the same temperature and pressure contain the same number of molecules Therefore, a gram molecular weight of any gas at the same temperature and pressure occupies the same volume At standard conditions (0 C and 760 mm Hg pressure) this volume is 22 4 liters At room temperature it is approximately 24 liters

Density of a Gas It is expressed as the weight in grams of one liter of the gas at standard conditions

Specific Gravity The weight of a volume of the gas compared to the weight of an equal volume of air under the same conditions

Critical Temperature That temperature above which the gas cannot be liquified regardless of the pressure applied

Critical Pressure That pressure below which the gas cannot be liquified regardless of the temperature to which it is cooled

Diffusion The process whereby gas molecules distribute themselves quickly in a space so that they exert equal pressures on all parts of the walls limiting the space A gas diffuses from a place of high partial pressure to that of lower partial pressure

Graham s Law The rate of diffusion of gases varies inversely as the square roots of their molecular weights Therefore heavier gases diffuse more slowly

Boiling Point The temperature at which the vapor pressure of a liquid equals atmospheric pressure

Vapor Many loosely define a vapor as the gaseous state of a substance which at room temperature and at-

XI

PHYSICS — ANESTHETIC GASES AND VAPORS

THE ANESTHETIC gases and vapors have physical and pharmacological properties which vary one from the other These properties make for advantages and disadvantages which are of minor importance in the average good risk patient, but in the poor risk patient the choice of agent is of greater significance The well trained anesthesiologist should be familiar with the properties of all the agents to be able to choose the best agent and technique for a particular operation on a particular patient

PHYSICS OF VAPORS AND GASES

Gas Refers to the state of a substance in which the molecules are in rapid motion and widely separated It is capable of infinite expansion and can be converted to a liquid by pressure and/or lowering the temperature The substance is usually in the gaseous state under ordinary conditions of temperature and pressure

Pressure Refers to the force exerted by the molecules of the gas constantly bombarding the walls of its container

Boyle s Law The volume of a gas varies inversely as the pressure, when the temperature remains constant

Charles Law The volume of a gas is directly proportional to its absolute temperature, when the pressure remains constant For each degree of temperature change the volume will change $1/273$

circulation, a considerable fresh supply of ether must be inspired to keep the blood (and brain) high in ether concentration This means that induction will be a relatively slow process During recovery, for each 16 molecules brought to the lungs only one will pass into the alveoli resulting in a slow recovery

Respiration is stimulated during light anesthesia Blood pressure falls in deep anesthesia In elderly patients and those with myocardial disease this blood pressure fall may be seen even in light anesthesia The intestines are dilated, muscle relaxation is good Bronchiolar dilatation occurs so that this is a good agent for asthmatics

CYCLOPROPANE Maintenance concentration is about 15 to 25% in the inspired air, with a blood level of about 16 to 20 mg % Blood/air ratio is 0 4/1 Therefore, one may expect a fast induction and recovery This agent is much less irritating than ether but high concentrations during induction may be irritating enough to cause laryn gospasm Rate and depth of respiration are reduced significantly so that assistance of respiration by the anesthesiologist is frequently necessary to maintain an adequate exchange Bronchiolar constriction may occur so that this drug must be used with caution in asthmatics Arrhythmias may occur in any plane, but are most common in deep planes during hypoventilation Epinephrine is contraindicated due to increased cardiac irritability Peripheral circulation is well maintained making this drug an agent of choice in hemorrhage Postoperatively there may be hypotension which readily responds to treatment with vasopressors

NITROUS OXIDE Blood/air ratio 0 3/1

ETHYLENE Blood/air ratio 1 2/1

Both are very fast acting agents for induction and recovery Of all the anesthetic agents these are the least

mospheric pressure can also be in the liquid state To be more exact below the critical temperature, which varies for each substance, the molecules are called vapors since they are or could be in equilibrium with the same molecular species in the liquid state The concentration of the vapor molecules in equilibrium with liquid would depend entirely on the temperature provided the liquid with which it is in equilibrium is pure Above the critical temperature, no liquid may exist, and the molecules are then called gases, rather than vapors The concentration of gas molecules depends on other conditions besides temperature Any change in pressure and/or volume of a vapor does not change the vapor concentration (unlike a gas) provided there is no temperature change and the vapor is in equilibrium with its liquid phase

The anesthetic vapors and gases available are

1 Di ethyl ether (common ether)
2 Cyclopropane
3 Nitrous oxide
4 Ethylene
5 Chloroform
6 Ethyl chloride
7 Trichlorethylene (Trilene)
8 Di vinyl ether (Vinethene)

Di ethyl Ether Induction requires 10 to 12% in the inspired air Maintenance requires about 4 to 6% which results in a blood concentration of 110 to 140 mg % The blood/air ratio is 15/1 indicating that ether is relatively much more soluble in blood than the other gases and vapors At equilibrium blood contains 15 molecules for each molecule in the alveolar air During induction, for each 16 molecules inspired, 15 go into the blood and only one remains in the alveoli With the next pulmonary

used in a non-rebreathing technique Muscular relaxation is poor when used in safe planes of anesthesia

DI-VINYL ETHER Induction requires about 4% in the inspired air The anesthetic blood level is 15 to 30 mg % This drug produces a very fast induction and recovery is also speedy It is a potent drug and respiratory arrest is quickly attained It is usually used by open drop procedure for short cases or for inductions before ethyl ether in children Increased tidal volume and salivation are frequent Twitchings and convulsions have been reported especially if the administration of the drug is associated with hypoxia

TABLE III

Drug	*Mol Wt*	*Spec Grav (Air = 1)*	*Boil Pt (C)*	*Flammability*	*Usual Cylinder*		
					Size	*Gal*	*Pressure (lbs / in²)*
Ethyl ether	74	2 6	36	+			
Cyclopropane	42	1 5	−33	+	B	100	75
Nitrous Oxide	44	1 5	−89	0	D	250	800
Ethylene	28	0 97	−105	+	E	330	1250
Chloroform	119	4 1	61	0			
Ethyl Chloride	64	2 3	12 5	+			
Trichlorethylene	130	1 5	87	0			
Vinyl ether	70	2 2	28	+			
Helium	4	0 18		0	E	131	1600
Oxygen	32	1 1		0	D	95	1800

potent, ethylene being a little more potent than nitrous oxide They usually require an additional reinforcing agent to reach surgical anesthesia Both agents must always be given in such a way that at least 20% oxygen is delivered to the patient These two agents have no toxic effects if not given in hypoxic mixtures Muscular relaxation is very poor

CHLOROFORM Induction requires about 4% in the inspired air, maintenance about 1% During surgical anesthesia the blood level is 7 to 13 mg % This drug is relatively nonirritating to the respiratory tract Overdose is common because the margin of safety is narrow Bradycardia occurs with overdose and is an indication to stop the administration Cardiac output and blood pressure are reduced due to direct myocardial depression Ventricular fibrillation or hepatic degeneration may occur if hypoxia is present Muscular relaxation is good Induction is smooth, relatively fast although recovery may be prolonged It is an excellent analgesic for short cases

ETHYL CHLORIDE For anesthesia 2 to 3% concentration of the inhaled vapor is necessary The blood concentration during anesthesia is 20 to 30 mg % It is slightly less toxic and slightly less potent than chloroform It also depresses the myocardium and the blood pressure Hyperventilation occurs in light levels Induction and recovery are fast It is only used for short cases or for open drop induction in children before adding ether

TRICHLORETHYLENE A concentration of 0 5 to 1 0% is sufficient for first plane anesthesia If used for deeper anesthesia tachypnea develops Both tachycardia and bradycardia may occur On contact with alkali present in CO_2 absorption canisters it decomposes to di chlor acetylene which is highly toxic Therefore, it can only be

thiopental Oxygen concentrations are never less than 20%

Thiopental may be used for induction prior to the use of inhalation anesthesia In these cases 2 or 3 cc doses of 2½% thiopental are given every 30 to 60 seconds until the patient falls asleep and does not object to the placing of the mask on the face When thiopental is to be used throughout the operation induction is performed in the same manner, and small increments (1 3 cc) are added intermittently to reach and maintain surgical anesthesia Early in the procedure relatively more thiopental is needed per unit of time because the drug is distributed rapidly to body fat[1] When fat storage is satisfied plasma levels can be maintained more easily with smaller doses If large total doses are used the patient may remain unconscious for a long period About 10 to 15% of thiopental is metabolized per hour Total doses over 1 5 to 2 0 grams are not recommended

During long surgical procedures it has become common practice to supplement thiopental nitrous oxide oxygen anesthesia with Demerol® or morphine, to avoid the administration of large quantities of thiopental Demerol® is given intravenously in doses of 10 to 25 mgm when there are signs of lightening of anesthetic depth Morphine is used in doses of 1 to 3 mgm A good index to follow is the respiratory rate Small narcotic doses are given until the rate drops to about 12 per minute and subsequent doses added when the rate rises to about 18 per minute Most patients managed in this way will awaken within 5 to 20 minutes after nitrous oxide is discontinued

Respiratory Effects Thiopental depresses the respiratory center and causes a reduction of rate and depth of respiration Overdose can, therefore, result in hypoventilation and apnea Therapy consists of assisted or artificial

XII

INTRAVENOUS ANESTHESIA

INTRAVENOUS ANESTHETICS afford a deceptively simple means of producing and maintaining general anesthesia Induction is rapid and pleasant for the patient When a nonflammable technique is required, intravenous anesthesia supplemented with nitrous oxide oxygen inhalation may be used The thiobarbiturates, sodium Pentothal® (thiopental) or sodium Surital® (thiosecobarbital) are usually used, and sodium Nembutal® (pentobarbital) or sodium Seconal® (secobarbital) less frequently

Thiopental Sodium This is probably the most commonly used intravenous general anesthetic It is available in powder form which is diluted with sterile water or normal saline solution The usual concentrations employed are 2½% when intermittent doses of drug are injected, or 0 1 to 0 5% when a continuous drip is administered Each gram of thiopental is buffered with 60 mgms of sodium carbonate so that the resulting solutions are alkaline (2½% solution has a pH of 10 to 10 5) Drug precipitation occurs when acid solutions (Demerol® curare) are injected into the same tubing used for thiopental

Sleep and depression of respiration occur before there is marked diminution in the reflex response to pain The use of thiopental as the sole agent for surgical anesthesia is hazardous because of hypoventilation and circulatory depression that will result It is therefore, common practice to use nitrous oxide oxygen or ethylene oxygen with

dren tolerate more (80-100 mgm /kgm) Avertin® is diluted with warm water (104 F) to a 2 5% solution and tested with Congo Red If the Congo Red turns the solution purple it indicates that the tribromethanol has decomposed into hydrobromic acid, tribromacetaldehyde, and other products which will irritate the tissues and cause proctitis A pink color indicates that no decomposition has taken place and the solution may be used

Hypnosis begins in about 5 minutes, reaches a peak in about 25 minutes, and lasts from about 1 to 1½ hours It takes several hours for complete consciousness to return When supplemented with nitrous oxide it is a good nonflammable method for a procedure of moderate duration

Respiratory Effects Avertin® causes depression of rate and depth of respiration In some patients this will necessitate assistance to respiration to maintain efficient gas exchange in the lungs Laryngeal reflexes are diminished and bronchodilation occurs Therefore, it is a good drug for bronchial asthmatics

Circulatory Effects The blood pressure falls about 20 to 30% following the administration of the drug This usually rises to normal when operation is begun If hypotension persists or systolic pressure drops below 80 mm Hg a vasopressor is indicated Ephedrine 12 to 15 mgm intravenously and 35 mgm intramuscularly provide effective treatment

Detoxification The drug is detoxified by the liver and the products excreted in the urine Some degree of reversible liver damage (central necrosis) may occur It is therefore not recommended for patients with liver or kidney damage, nor wise to repeat this drug in less than one week

respiration Thiopental enhances the laryngeal reflexes and may cause broncho constriction It is, therefore, not recommended during attacks of bronchial asthma

Cardiovascular Effects A small degree of blood pressure fall usually follows the injection of thiopental In patients with deficient blood volumes or poor cardiovascular reserve hypotension may be profound It is thought to be due to depression of the vasomotor center with resultant vasodilatation

Muscular Relaxation Thiopental is a poor muscular relaxant To obtain relaxation a potent general anesthetic agent or a muscle relaxant must be added

RECTAL ANESTHESIA

Anesthetic drugs administered rectally are usually used to produce basal anesthesia (hypnosis) and therefore require some supplementary form of anesthesia The usual drugs used are sodium thiopental or Avertin® A cleansing enema prior to operation permits better absorption of these drugs

Sodium Thiopental Administered as a 5% solution The dosage is 15 to 20 mgm per pound of body weight It will begin to produce the hypnotic effect about 10 to 15 minutes after administration and the maximum effect is reached in 30 minutes The duration of significant effect will be approximately 1 hour but it takes several hours for complete consciousness to return Systemic effects are the same after rectal instillation as after intravenous injection

Avertin® A solution of tribromethanol with amylene hydrate The latter serves as the solvent but does have mild hypnotic properties One cc of Avertin® contains 1 gm of tribromethanol and 0 5 gm of amylene hydrate In adults the average dose is 50 to 80 mgm /kgm Chil

XIII

NEUROMUSCULAR BLOCKADE AND ITS REVERSAL

A BRIEF DESCRIPTION OF DEPOLARIZATION

A COMMON PROPERTY of all living cells is the existence of an electrical potential across the cell membrane, the inside of the cell being approximately one tenth of a volt negative with respect to the extracellular fluid. An important factor contributing to the generation of this potential is the existence of high concentration gradients of several important electrolytes across the membrane. The concentration of potassium within the cell is approximately 20 times that of the extracellular water. On the other hand, the concentration of sodium within the cell is one sixth of that of the extracellular water. There is a slow exchange of these cations across the membrane during the resting state. When an excitable cell such as a nerve or muscle fiber is depolarized several changes occur which are characteristic of the event. The resting potential first collapses (depolarization) then proceeds to reverse itself slightly and finally returns to its original state (repolarization). During depolarization, the rate of exchange of potassium and sodium ions is markedly increased. The sodium ions enter the cell twenty times faster than potassium leaves the cell. There is evidence to indicate that the change of electrical potential across a membrane is the result of migration of these cations and a decrease of cationic concentration gradients across the membrane. The movement of cations across the de-

Since the drug is administered rectally, proctitis, colitis or colonic hemorrhage are contraindications

REFERENCE

1 Brodie, B B, Bernstein, E and Mark, L M The role of body fat in limiting the duration of action of thiopental *J Pharmacol & Exper Therap* *105* 421 426 1952

tered enough to produce a widespread depolarization of the muscle fiber (d-tubocurarine, dimethyl d tubocurarine, Flaxedil®, and Mytolon® belong to this group) Anticholinesterases (Tensilon®, prostigmine) antagonize the action of these agents by allowing the concentration of acetylcholine to build up to overcome the neuromuscular blockade The other group of agents affect neuromuscular transmission by depolarizing the motor end plate so that it becomes refractory to motor impulses Decamethonium and succinylcholine belong to this group During the early phase of depolarization, the excitability of the end plate is actually increased before it passes into the state of refractoriness The paralysis is usually preceded by a period of muscular fasciculations

However, such a classification of relaxants only indicates the main pharmacological property of each agent Indeed, with various concentrations it was demonstrated that many of these agents have multiple sites and patterns of action A thorough familiarity with the pharmacological and physiological effects of each agent is essential for the intelligent and safe usage of these drugs

d Tubocurarine The average paralyzing dose for the average adult is 6 to 12 mgms The muscular paralysis lasts from 30 to 45 minutes The effect of repeated administration is cumulative Ether potentiates its activity and when ether anesthesia is used this drug should be given with caution and the dosage reduced to one third or one half Although the respiratory muscles are the last to be paralyzed, with full paralyzing dose these muscles are not spared Clinical paralyzing doses do not cross the placenta in sufficient quantity to endanger the newborn [1] The use of this drug in patients with myasthenia gravis is contraindicated Occasionally, bronchoconstriction can be caused by d tubocurarine [2]

polarized part of the cell membrane further serves to depolarize the adjacent membrane and in this manner the impulse is propagated along the nerve or muscle fiber Repolarization is accomplished by active expulsion of sodium ion and restoration of the cationic concentration gradient, accompanied by evolution of heat, increased uptake of oxygen, and glucose utilization

A BRIEF ACCOUNT OF NEUROMUSCULAR TRANSMISSION

The nerve impulses are conducted along the motor nerve from the spinal cord as transient waves of depolarization Concurrent with its arrival at the nerve end plate there is a discharge of acetylcholine A reaction occurs involving acetylcholine and receptor substances located in the end plate region of the muscle fiber As a result of this reaction, a localized depolarization occurs in the end plate region If this depolarization is of sufficient magnitude there results a spreading, self sustained depolarization of the muscle fiber, accompanied by excitation of the contraction process of the myofibrils Acetylcholine is removed from the end plate region almost as fast as it is formed by cholinesterase and by resynthesis of acetylcholine precursors During the depolarized period the muscle is refractory to further stimulation

TYPES OF NEUROMUSCULAR BLOCKADE

The currently popular relaxant drugs fall into two groups depending upon their mode of action in producing neuromuscular blockade One group of agents blocks neuromuscular transmission by interfering with normal activity of the acetylcholine released at the motor nerve endings These agents stabilize the motor end plate and prevent depolarization The end plate potential is not al-

be sudden Its effect is not cumulative and tachyphylaxis can occur to make the effect of repeated injections unpredictable The respiratory paralyzing effect is more marked than that of d-tubocurarine There is no evidence of histamine release It does not cause bronchiolar spasm and hypotension, nor does it cross the placental barrier[3, 4]

Succinylcholine It is a depolarizing agent The duration of its action is extremely short Following a single injection of 20 to 60 mgms (the average dose) the paralysis may last from 5 to 7 minutes For sustained relaxation, it is given as a continuous infusion in 0 1 to 0 2% solution The degree of relaxation can be adjusted by regulating the rate of infusion It is remarkably free from side effects although hypertension can occasionally occur The hypertension is probably due to its nicotinic action on the sympathetic ganglia[9]

Succinylcholine is hydrolyzed in the body by pseudo cholinesterase The dose and duration of paralysis have a direct relationship There is neither cumulative effect nor tachyphylaxis In patients with hepatic damage, in whom the plasma pseudo cholinesterase level is low, the duration of action can be greatly prolonged[10] Anticholinesterases markedly potentiate the effect of succinyl choline[11]

Relaxants usually require endotracheal methods In any event, they must never be used without endotracheal equipment, facilities for artificial ventilation, and skill sufficient to intubate and ventilate

Antagonists to Relaxants

Prolonged paralysis and apnea can occur after the administration of relaxants If there is spontaneous respiration the ventilatory effort may be totally inadequate for the gas exchange This complication is not related to any

Dimethyl d tubocurarine, Metubine® The paralyzing dose for the average adult is 1 5 to 3 0 mgms Its duration of action is slightly shorter than that of d tubocurarine It has a higher coefficient of safety with regard to the respiratory sparing effect as measured by the ratio of vital *capacity depression and grip strength depression* [3, 4]

Flaxedil®, Gallamine The paralyzing dose for the average adult is 40 to 60 mgm Its potency is one fourth that of d tubocurarine (mgms for mgms) The peak action is reached in 5 to 6 minutes and the duration of its effect is shorter than that of d-tubocurarine Cumulative action has been demonstrated It does not liberate histamine-like substances and with clinical paralyzing doses it does not block ganglionic transmission However, it has a specific vagolytic action on the heart, resulting in tachycardia which lasts for 20 to 30 minutes Because of these side actions it should not be used in patients with significant cardiovascular disturbances [5, 6]

Mytolon® Its action at the neuromuscular junction is similar to that of d tubocurarine The paralyzing dose for the average adult is 6 mgms Its peak action occurs in 6 to 8 minutes The recovery from paralysis is gradual Its action at the neuromuscular junction is similar to that of d tubocurarine but it also has an anticholinesterase activity It potentiates the vasodepressor response following vagal stimulation and the action of acetylcholine Because of the anticholinesterase activity excessive mucus secretion and sinus bradycardia occur quite often Adequate premedication with belladonna drugs prevent the occurrence of these side effects [7, 8]

Decamethonium, Syncurine® The average initial dose is 1 to 4 mgms Its effects reach the maximum in 4 to 6 minutes The duration of action is shorter than that of the agents discussed above Recovery from paralysis can

in two thirds of the patients who receive this drug, and bronchiolar spasm and bronchorrhea occur occasionally [14]

There is no antagonist to decamethonium nor succinylcholine available for clinical use

REFERENCES

1 Buller A J and Young, J Action of d tubocurarine chloride on fetal neuromuscular transmission and placental transfer of this drug in rabbits *J Physiol*, *108* 412 420, 1949

2 Landmesser C M, Converse, J G and Harmel, M H Quantitative evaluation of bronchoconstrictor action of curare in anesthetized patients *Anesthesiology*, 13 275 280, 1952

3 Unna, K R, *et al* Evaluation of curarizing drugs in man *J Pharmacol & Exper Therap* *98* 318 329, 1950

4 Pelikan E W, *et al* Evaluation of curarizing drugs in man *J Pharmacol & Exper Therap*, *99* 215 225, 1950

5 Unna K R *et al* Evaluation of curarizing drugs in man *J Pharmacol & Exper Therap*, *100* 201 209, 1950

6 Marbury, B E, *et al* Effects of synthetic curare like compound Flaxedil on the anesthetized surgical patient *J Pharmacol & Exper Therap* *103* 280 287 1951

7 Hoppe J O Pharmacological investigation of Win 2747 new curarimimetic drug *J Pharmacol & Exper Therap* *100* 333 345 1950

8 Robertazzi R W, Discussion of Foldes F F Use of Mytolon chloride in anesthesiology *Ann New York Acad Sc*, *54* 503-511 1951

9 Thesleff S Pharmacological properties of succinylcholine iodide, with particular reference to its clinical use as a muscular relaxant *Acta physiol scandinav*, *26* 103 129 1952

particular relaxant nor is it more likely to occur with one anesthetic agent than another It is dangerous and may be fatal if the condition is not treated Careful and judicious use of the relaxants can help a great deal to avoid these complications If they occur the patient should be kept under supervision and artifically ventilated until he has completely recovered from the effects of relaxants Antagonists to relaxants may be useful, but as the duration of these drugs is relatively transient, its action should not be relied upon for the management of prolonged paralysis

The "depolarizing blocking agents and depolarizing agents are mutually antagonistic In experimental animals d tubocurarine has an antagnostic action against decamethonium and suitable doses of decamethonium also restores the neuromuscular transmission blocked by d tubocurarine However, this pharmacological phenomenon is not applicable clinically

Anticholinesterases, such as Tensilon® and prostigmine, are antagonistic to relaxants with depolarization blocking action The effect of prostigmine is transient and side reactions, such as bronchorrhea and bronchiolar spasm are common [12] Belladonna drugs can be used to minimize these side effects

Tensilon® a phenolic quarternary ammonium derivative is a more important antagonist for d tubocurarine and similar agents It has been shown to have anticholinesterase activity which is thought to be responsible for its activity by some investigators [12] Others have demonstrated that Tensilon® antagonizes curare at the myo neural junction by its direct effect on the motor end plate and its competitive action against curare [13] In doses of 5 to 15 mgms its action appears within 1 minute and lasts for about 15 minutes Transient bradycardia occurs

XIV

COMPLICATIONS OF GENERAL ANESTHESIA

THE POSSIBLE complications of general anesthesia are many For convenience these will be discussed in four critiques respiratory, circulatory, neurological and miscellaneous

I RESPIRATORY COMPLICATIONS

A *Obstruction of the Airway* Obstruction is defined as mechanical interference with the proper exchange of gases (oxygen and CO_2) between the external environment and the alveoli It is the most frequent complication of general anesthesia Complete obstruction, if not remedied within a few minutes, can be fatal Partial obstruction is also harmful especially in a debilitated patient or one with a poor cardiovascular reserve The signs of obstruction of the airway include increased effort of the respiratory muscles associated with the movement of a small amount of gas noisy respirations and cyanosis Although noisy respirations indicate that some obstruction is present, not all types of obstruction need necessarily be noisy

1 LIPS may cause obstruction in edentulous patients and in children with enlarged adenoids Therapy is the insertion of an orophyngeal airway

2 TONGUE AND PHARYNX relax and block the glottic opening in the supine position When the tongue falls

10 Bourne J G Collier, H O J and Somers G F Succinylcholine muscle relaxant of short action *Lancet* *1* 1225 1229 1952

11 Castillo J C and de Beer, E J Neuromuscular blocking action of succinylcholine (diacetylcholine) *J Pharmacol & Exper Therap* 99 458-464, 1950

12 Smith, C M, *et al* Mode of action of antagonists to curare *J Pharmacol & Exper Therap* *105* 391-399 1952

13 Wescoe, W C and Riker, W F Pharmacology of anti curare agents *Ann New York Acad Sc*, *54* 438 549, 1951

14 Artusio J F Riker, W F and Wescoe W C Studies on inter relationship of certain cholinergic compounds *J Pharmacol & Exper Therap* *100* 227 237 1950

the respiratory from the gastro intestinal tracts General anesthesia can then be induced with little danger should vomiting occur Separation of the airway and the gastro intestinal tract can be achieved by intubating rapidly during apnea induced by a muscle relaxant Vomiting cannot occur and, if the semi recumbent posture is used, regurgitation is unlikely Intubation by both methods is for the experienced anesthesiologist It should not be attempted by the beginner without expert guidance Silent regurgitation of stomach contents may occur during induction or maintenance The only sign of this complication may be the cessation of respiration at a time when no increase in the concentration of an irritating vapor nor insertion of an airway has taken place Attempts to inflate the chest are usually futile until the irritating gastric contents have been suctioned out of the oropharynx

Should vomiting or regurgitation occur, the patient is placed in Trendelenburg position at once The oro- and nasopharynx are suctioned as quickly as possible The trachea is intubated if necessary and sucked out Rarely, bronchoscopy may be necessary No general anesthesia should be begun without suction apparatus in working condition and directly at hand

6 Tumors in the Oropharynx—Deviated or Compressed Trachea Endotracheal intubation with topical anesthesia is indicated

7 Laryngospasm Partial or complete adduction of the vocal cords occurs frequently Partial laryngospasm produces a high pitched crowing noise

Causes

a) foreign body at the vocal cords (blood, mucus, vomitus improperly placed oropharyngeal airway)

b) Irritating gases and vapors

back a snoring noise is produced Therapy consists of pulling the mandible forward and the insertion of an oropharyngeal airway

3 FOREIGN BODIES Chewing gum, loose teeth, and blood clots may obstruct the glottis To prevent this complication be *sure all dentures are removed* and the oropharynx is empty before proceeding with anesthesia

4 EXCESSIVE MUCUS can be prevented by adequate belladonna premedication and continuous maintenance of unobstructed airway Obstruction tends to produce mucus and mucus produces further obstruction with progressively increasing respiratory difficulty Suction aspiration of the mucus from the pharynx or trachea is necessary *Ether frequently causes excessive mucus secretion* in light planes of anesthesia Deepening the anesthesia may be an effective remedy

5 VOMITING is a serious problem because of the possibility of aspiration of foreign material into the airway Pneumonitis atelectasis and total airway obstruction are possible consequences of this accident Food may be retained in the stomach for many hours when there is *pain or apprehension This situation is particularly pertinent* in emergency surgery for trauma and in obstetrics Elective patients should fast for 8 to 12 hours preoperatively A smooth induction and avoidance of uneven light anesthesia diminish the incidence of vomiting In emergency surgery, food in the stomach is always suspected Passage of a gastric tube and lavage help to empty the stomach This procedure is not absolutely secure since a part of the gastric contents can remain Inducing vomiting before anesthesia also is not a sure way A safer method, when general anesthesia is used is the performance of endotracheal intubation with a cuffed tube with topical anesthesia while the patient is awake to isolate

due to the high negative intrapulmonary pressure produced during attempted inspiration This complication is rare except in patients with diseased hearts and in infants and children Obstruction must be relieved as part of the therapy

B Anoxia or Hypoxia

CAUSES

1 *Respiratory obstruction* (see above)

2 *Decreased oxygen tension in the inspired air* which may be due to a wrong tank on the oxygen yoke, failure to recognize the emptying of the oxygen tank or an open N_2O or ethylene flush valve Always check the machine to see that the tanks are not empty, are in their proper place and that all valves are shut before starting anesthesia A good way to avoid placing a wrong tank on a yoke is never to change more than one tank at a time

3 *Overdosage of depressant drugs* so that the respiratory effort is insufficient to bring enough O_2 to the alveoli The extreme is respiratory arrest Therapy consists of assisting the respiration by squeezing the bag during inspiration and lightening the anesthesia

4 *Restraint of thoraco diaphragmatic movement* due to surgical assistants leaning on the abdomen or chest, due to too many packs in the abdomen, due to steep Trendelenburg or lateral kidney position, due to gastrointestinal distention Therapy consists of removing the cause and assisting the patient s respiration

C CO_2 Excess

CAUSES

1 *Respiratory obstruction* (see above)

2 *Soda lime exhausted* in canister

3 *Dead space* out of proportion to tidal volume so

c) Reflex laryngospasm caused by sudden visceral traction or skin incision before the establishment of adequate depths of anesthesia

Therapy

a) Remove the cause if possible, i e , suction out blood, *vomitus, or mucus, lower concentration of irritating gas,* deepen anesthesia if due to reflexes

b) Pressure on breathing bag, especially during expiration with the chin pulled well forward

c) Muscle relaxant injected intravenously in small doses

d) Endotracheal intubation

e) Tracheostomy if above measures fail and circulation is failing

8 Bronchiolar Spasm Patients with asthma or other allergic manifestations are prone to develop this complication The patient usually develops wheezes on expiration and expiration occurs with increasing difficulty

The causes are similar to those of laryngospasm

Therapy consists in removing the cause if possible, changing the agent—ether is especially good, succinylcholine has been of real help and much safer than d tubocurarine since bronchiolar spasm can be produced by d tubocurarine itself Aminophylline is ineffective and may cause circulatory collapse and death in the anesthetized patient Hydrocortisone intravenously offers the possibility of effective therapy without side effects

A Consequences of Respiratory Obstruction

1 Asphyxia A combination of O_2 lack and CO_2 excess This can lead to circulatory failure and cardiac arrest

2 Pulmonary Edema which is usually a transudate

3. Teeth
4. Sponges and packs

Therapy consists of removal of foreign bodies, adequate suction, use of the Trendelenburg position, endotracheal suction, making the patient cough, and finally bronchoscopy if necessary.

SEQUELAE—ATELECTASIS

This is due to the plugging of a bronchus and the absorption of air distally. Incidence with upper abdominal and kidney operations is about 6%. Five tenths per cent of all patients operated on show some partial atelectasis. Twenty five per cent of patients with massive atelectasis die. This complication can lead to pneumonia or a lung abscess.

Other causes of atelectasis besides aspiration during and after anesthesia include:

a) Depressed ciliary activity and cough reflex so that mucus can accumulate and form a plug in a bronchus.

b) Depressed respiration restricting pulmonary ventilation.

c) Too long an endotracheal tube will usually pass into right bronchus and cause atelectasis of left lung.

d) External pressure, as in steep Trendelenburg or kidney position.

SIGNS AND SYMPTOMS

a) Assymetry of chest movement
b) Rib spaces narrowed on affected side
c) Tracheal shift to side of collapse
d) Breath sounds diminished or absent
e) Dullness to percussion
f) Signs of oxygen want—fast pulse, increase respiratory rate and fever. The last sign is seen later in the postoperative period.

that excessive rebreathing occurs An example is the use of a large mask in a closed system for children

4 *Exhalation valves* on machine defective

D Apnea Burstein[1] differentiates this from respiratory arrest due to overdose In apnea there is a sudden cessation of respiration

CAUSES

1 Complete respiratory obstruction

2 Breath holding during induction

3 Reflex as seen in periosteal irritation, tugging on the mesentery or irritation of the diaphragm

4 Carotid body mechanism With a depressed medullary respiratory center, carbon dioxide cannot stimulate respiration adequately The resulting hypoxia will reflexly cause respiratory activity by stimulation of the chemoreceptors in the carotid and aortic bodies If such a patient is then placed on an inspiratory mixture containing a high oxygen concentration so that the hypoxia and thereby the stimulus to the chemoreceptors is eliminated, apnea will result Therapy consists of assisted or controlled respiration with adequate oxygen until the depressant drugs have been metabolized and the respiratory center can function properly

5 Hyperventilation and excessive elimination of CO_2 due to removal of carbon dioxide stimulation to respiration

6 Pressure on carotid sinus or vagus nerve stimulation

7 Controlled respiration—artificially inducing apnea by hyperventilation

E Aspiration

1 Vomitus Most frequent cause

2 Blood clots and pus if operating in oropharynx

A Tachycardia

CAUSES

1) Excitement in the second stage
2) Light anesthesia with painful stimuli
3) Blood loss
4) Thyrotoxicosis
5) Use of epinephrine in surgical field to control bleeding
6) Intravenous atropine—seen especially in children
7) Asphyxia—early
8) Improper controlled respiration so that there is intereference with venous return to the right heart and with pulmonary circulation
9) Adrenal cortical insufficiency and pheochromo cytoma
10) Myocardial infarct
11) Hyperpyrexia
12) Paroxysmal auricular tachycardia

B Bradycardia

CAUSES

1) Asphyxia—late
2) Vagal stimulation, e g neck operations with hyperactive carotid sinus reflex, pressure on eye during light anesthesia
3) Cyclopropane anesthesia
4) Certain vasopressors (neosynephrine or norepinephrine)
5) Increased intracranial pressure

C Hypertension

CAUSES

1) CO_2 excess, asphyxia
2) Excitement in second stage

g) Cyanosis (depending on extent)

THERAPY—"Stir Up

a) Hyperventilate by rebreathing

*b) Turn frequently and encourage to cough

c) Pounding side of chest affected

d) Intercostal block to permit cough without pain

*e) Intratracheal suction

f) Bronchoscopy if needed

g) Oxygen for cyanosis or signs of oxygen lack

h) Antibiotics

*i) Cough machine"—exsufflation with negative pressure (Barach)

F Tachypnea Fast respirations are usually shallow and are fatiguing Because of the small tidal volume, the dead space assumes relatively larger proportions and therefore the actual gas exchange is poor

CAUSES

1) Light anesthesia

2) Inadequate premedication

3) Effect of agent stimulation on respiration especially ether

4) Anoxia including shock

5) Hyperpyrexia

THERAPY

Depending on cause deepen anesthesia if too light, assist respiration to ventilate patient better, control respiration intravenous morphine or Demerol® if cause is not anoxia or finally change agent

II CIRCULATORY COMPLICATIONS

These are manifested by alterations in rate and character of the pulse changes in blood pressure and rhythm of the heart

* Stars indicate more important therapeutic procedures

blood and digitalization intravenously Positive pressure O_2 is useful

E Arrhythmias

CAUSES

1) Asphyxia

2) Epinephrine used in the presence of cyclopropane, chloroform, or trichlorethylene

3) Effect of agent (cyclopropane)

4) Thyrotoxicosis

5) Following endotracheal intubation

6) Reflex from surgical field

F Cardiac Arrest

III NEUROLOGICAL COMPLICATIONS

A Convulsions

1) Anoxia and excessive CO

2) Ether convulsions The cause is unknown Possible contributing factors are excessive CO_2, hypoxia toxemia, hyperthermia dehydration and acidosis It is usually seen in a toxic dehydrated child with a high temperature It can occur at any time and at all depths of anesthesia Twenty per cent die from asphyxia and circulatory failure Therapy should be directed towards stopping the convulsions and supplying oxygen in high concentration Intravenous barbiturates and artificial respiration with 100% oxygen are efficient therapy

B Emergence delirium is a stage of excitement during recovery from anesthesia It is seen most frequently following the use of cyclopropane

THERAPY

a) Sedation with intravenous morphine or Demerol® especially if there is pain

3) Light anesthesia with stimulation
4) Epinephrine in surgical field
5) Excessive vasopressor use
6) Thyrotoxicosis
7) Pheochromocytoma
8) Hyperthermia
9) Increased intracranial pressure

D Hypotension

CAUSES

1) Blood loss

2) Effect of agent on heart muscle or vasomotor center

3) Sudden change in position during general anesthesia The compensatory circulatory reflexes are depressed Moving the patient suddenly will frenquently result in hypotension

4) Asphyxia—late

5) Post cyclopropane especially if respiration has become depressed and is not assisted to prevent an accumulation of CO_2 Treatment with a vasopressor is indicated

6) Improper controlled respiration

7) Reflex—secondary to pulling mesentery or working on bone

8) Coeliac plexus reflex During upper abdominal exploration this reflex will produce a drop in systolic but not diastolic blood pressure so that a very narrow pulse pressure results There is usually little change in pulse rate

9) Adrenal cortical insufficiency

10) Myocardial infarct or cardiac decompensation Overloading the circulation with fluid or blood will cause even a healthy heart to decompensate The first sign is that of pulmonary edema Therapy consists of removing

XV

SOME PRINCIPLES OF PEDIATRIC ANESTHESIA

THE REALIZATION that infants and children cannot be treated anesthetically as diminutive adults has resulted in the development of the subspecialty of pediatric anesthesia Awareness of the differences between children and adults in anatomy, physiology, and psychology, and the special hazards and complications most frequently encountered is imperative for proper intelligent management of pediatric anesthesia and fluid replacement therapy

Important Differentiating Features of Infants and Children (Infants can be considered those under 2 years of age and children from 2 to 12 years)

ANATOMICALLY the neck and chest have poor musculature the bony thorax is elastic and unstable and diaphragmatic activity predominates Consequently, part of the chest (especially the lower one third where the diaphragm is attached to the ribs) or all of the chest in young infants will retract during inspiration Therefore, optimum respiratory exchange is diminished since anteroposterior lung expansion is minimized In rapid or deep respiration when adequate air cannot enter the small larynx and trachea quickly enough this retraction of the intercostal spaces resembles respiratory obstruction and is confusing in determining the depth of anesthesia [1]

The abdomen of an infant is frequently large and bulky

b) Intravenous apomorphine, 1 to 2 mgms slowly until delirium stops or patient retches

IV MISCELLANEOUS COMPLICATIONS

A Fires and explosions

B Mask burns on face—most common on bridge of nose This is due to strapping on the mask too tightly To avoid, massage areas of pressure frequently and place wet cotton between mask and face

C Trauma to eyes from

1) Ether, which causes a conjunctivitis If liquid ether gets into a patient's eye, open the eye and blow to evaporate it, or irrigate with saline

2) Open eyes during anesthesia The cornea may be directly traumatized or may develop an ulcer from drying If the eyes are open pull the lid down and tape shut Never test for corneal reflexes

D Ether and chloroform burns to skin during open drop administration

E Broken teeth and cut lips

F Hiccough Treat with rebreathing to increase the CO_2 concentration, intravenous atropine, relaxant drugs controlled respirations deepening the anesthesia and phrenic nerve block

G Trauma to larynx during endotracheal intubation—may produce a torn vocal cord laryngeal edema, granuloma of cord

REFERENCE

1 Burstein C L *Fundamental Considerations in Anesthesia* New York, Macmillan 1949

Newborn to 1 year	— 0	
1 year to 2 years	— 1	
2 years to 3 years	— 2	
3 years to 4 years	— 3	
4 years to 5 years	— 4	
5 years to 9 years	— 5	(*Note* The size of the
9 years to 10 years	— 6	larynx changes little be-
10 years to 12 years	— 7	tween these ages)

Cuffed tubes are usually not necessary

The alveolar surface of the infant lung is relatively small and the dead space large This causes a large degree of rebreathing which decreases the efficiency of alveolar ventilation If one considers the small tidal volume of these patients the importance of the dead space becomes even more evident Shallow rapid respiration influences this factor even further As a result of the relatively larger anatomical dead space, any face piece that will increase dead space significantly will further magnify the effect of rebreathing Adult shaped masks are unsuitable Circuits with high resistance are unwise Recently the introduction of a childrens circle filter with a very small face mask and minimal dead space has proved practical Various techniques with nonrebreathing systems have also proved satisfactory especially in conjunction with endotracheal intubation to eliminate the dead space of the oropharynx Insufflation anesthesia using relatively large flows of gases reduces rebreathing problems The use of open drop anesthesia however, remains the most frequently used and most practical mode of general anesthesia in pediatrics Faulconer[4] showed that the oxygen content in the inspired atmosphere falls during the use of this technique and that the flow of 500 cc or more of oxygen under the mask will eliminate the hypoxia This

and can impede motion of the diaphragm and further depress respiratory exchange Associated with this is the peculiar ability of infants to swallow air[1] On inspiration a negative pressure occurs in the stomach and air is drawn in This ingestion is greater during obstruction to respiration and the stomach can become so distended that it seriously interferes with respiratory exchange A bulge in the left upper quadrant of the abdomen is usually visible and can be remedied by the insertion of a stomach tube to allow the gas to escape

The larynx of infants and children deserves special consideration It is more cephalad than in the adult, the epiglottis is relatively longer, stiffer, and more V shaped The cricoid ring is usually the narrowest portion of this part of the upper respiratory tract and the rima glottis is wider The result is a funnel shaped larynx Relative to the rate of gas flow through it, it is narrower than that of an adult This may also interfere with proper alveolar ventilation especially during deep breathing With the shape of the larynx in mind it is wiser to choose an endotracheal tube a size smaller than that which will pass through the glottis opening to avoid impingement at the cricoid constriction In choice of size of endotracheal tubes three sizes are recommended for preparation the size the anesthesiologist expects to use and one each of the next larger and the next smaller sizes These should be sterilized by heat and introduced without the use of a stillete, and without the use of a lubricant The possibility of laryngeal trauma and infection which can cause glottic edema are thereby minimized The use of a moist atmosphere for 12 to 24 hours postoperatively will also aid in reducing the incidence of laryngeal edema in infants and children who have been intubated The size tube recommended (Portex plastic) is

Newborn to 1 year	— 0	
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of hypoxia is poorly tolerated Recognition of respiratory obstruction is essential since immediate correction is mandatory

The heart rate of these patients also varies widely In infants anesthetized with ether it may run at 180 beats per minute In children a rate of 140 to 160 is not unusual[5] Apparently the vagal influence on the heart is less pronounced in infants than in adults A bradycardia (under 80 per minute) is a greater potential hazard than tachycardia[3]

The blood pressure of a newborn is about 80/46 mm Hg In two weeks it rises to 95/50 mm Hg and remains at this level during childhood[5] However, systolic blood pressure of 60 or 70 mm Hg is not uncommon nor cause for concern

Heat regulation by the infant is poor The temperature can fall to 92 F in a cool room or rise to 105 F in an overheated one or if too many drapes are used The tendency for hyperthermia is more common in infants and children over six months of age[3] If the temperature of the room is kept optimum (65 to 70 F), the drapes are kept at a minimum and air is blown over the child s body, the possibility of heat retention can be minimized It is probably better to maintain the temperature 1 or 2 F below normal than above If in doubt as to temperature change, rectal or esophageal temperatures should be taken to be certain

PREMEDICATION Children tolerate relatively large doses of premedicant drugs well In the past few years, the psychological effect of hospitalization and anesthetic induction in children have been emphasized These factors have encouraged the use of larger doses of premedicant drugs so that the child will not remember being brought to the operating room and the induction of anesthesia To

oxygen flow will also help remove accumulated carbon dioxide

The small body mass of these subjects deserves mention The blood stream and tissues can become saturated more rapidly with anesthetic agents[3] although this is compensated for, to an extent by the small tidal volume Once tissue saturation has been attained, overdose is very easy The smaller the child, the more apparent this is

PHYSIOLOGICALLY Changes in many functions in the young are rapid, variable and extreme because of relatively high metabolic activity and incomplete development of the central nervous system[2] Infants and children are more liable to generalized convulsions The rate of respiration can accelerate to 100 and abruptly slow to 20/min Irregular, gasping, or sobbing respirations are frequent and difficult to control These are usual in children who have cried strenuously before induction These types of respiration do not seem to be injurious though they may be annoying to the surgeon[1] Occasionally one side of the chest will move more than the other during inspiration In infants the respiratory rate averages 45 to 50 per minue[5] but variations from 20 to 100 are not unusual The tidal volume of a newborn is about 16 to 20 cc At 2 weeks of age it rises to 25 cc and continues to increase gradually thereafter It, therefore becomes obvious that very fast respiratory rates especially in infants are shallow with inefficient alveolar ventilation Methods of therapy of these *fast rates are intravenous morphine or meperidine with* controlled or assisted respiration

High oxygen demands by the infant and child require an uninterrupted oxygen supply The oxygen consumption of a newborn infant is approximately 7 cc per kg per minute (adults 3 9 cc /kg /min) and at 1 to 3 years of age may even be higher[5] Even a relatively short bout

amount of each drug is given 1 to 2 hours before anesthesia

The problem of preanesthetic fasting arises frequently in these young patients Infants feed approximately every 4 hours and since they have a high rate of metabolism, withholding the bottle longer not only makes them hungry and irritable, but also depletes their stores of glycogen[2] Therefore, about 4 hours before anesthesia, these infants should be given a bottle of orange juice or sweetened water Within 2 hours the stomach will be com pletely empty Older children need no food after supper, but can be given clear fluids (tea, water, soda) until about six hours before anesthesia

FLUID AND REPLACEMENT THERAPY Of all the phases of pediatric anesthesia, this one is handled the poorest Before it is presented a discussion of the principle of fluid balance in the infant and child is necessary

The unavoidable loss of fluid per day of an infant is about 4% of his body weight, which is proportionally twice that in an adult It is greater because of increased metabolism and because the infant kidney can only excrete a hypotonic urine Therefore, the infant can withstand water deprivation poorly[3] On the other hand, the kidneys of young patients have a very limited capacity of excreting excess fluid or sodium chloride[2] If one adds to this the fact that the kidneys will conserve salt if the intake is low, then it is seen that it is difficult to give too little salt The dangers of too much fluid and salt are greater than too little Excessive salt can produce peripheral, pulmo nary, and brain edema It is so easy to give intravenous fluids through a cut down that too much can be given It must also be remembered that during anesthesia kidney function is depressed[6]

Most pediatric surgery is extra abdominal and extra-

further assure a tranquil induction, pleasant techniques (such as blowing cyclopropane over the drowsy child until he can tolerate the mask for open drop ether, or the use of rectal Avertin® or thiopental) are indicated

The physiological age seems more important than the chronological age in determining dosage for premedication[1] The average infant weighing 7 pounds at birth will double that weight (14 pounds) at 5 months, triple it (21 pounds) at 1 year and approximately quadruple it (30 pounds) at 2 years After that a weight gain of 5 pounds per year of age is average

Premedication can be satisfactorily prescribed in a variety of ways Up to about 6 months of age, a drying agent such as scopolamine or atropine is all that is required A dose of 0 1 mgm will block secretions A sedative or narcotic is unnecessary in this young group In older infants and children various sedatives (secobarbital, pentobarbital) and narcotics (morphine, Demerol®) besides the belladonna drugs are needed Whether these drugs are used singly or in combination, they serve similar purposes The important factor is the dosage used

The usual dose of belladonna drug is 0 1 mgm until one year of age 0 2 mgm from 2 to 3 years of age, and 0 3 mgm for children over 4 years If only a barbiturate is added the usual dose recommended is 15 mgms for each year of age until about 4 or 5 years of age and about 10 mgms per year of age for 6 year olds or over If morphine is prescribed, the age minus one in mgm is given (i e a 6 year old would get 5 mgms of morphine) Meperidine can be used in doses 10 times that of morphine Many anesthesiologists prefer to use both a barbiturate and a narctoic When this is done the dosage is calculated the same as above and then two thirds of this

pound infant, etc If single intravenous infusions are given at intervals, the amount should not exceed 10 cc per pound and take 15 minutes to 1 hour to drop in depending on the size of the patient[2]

BLOOD TRANSFUSION The blood volume of an infant or child is approximately 10% of the body weight or 90 to 100 cc per kg of body weight An estimated blood loss in a 3 kg infant of over 30 cc (equivalent to 500 cc in an adult) should be replaced on the table This is about 8% to 10% of the estimated blood volume If the blood transfusion is for anemia without any significant blood loss during operation an amount equivalent to 10% of the estimated normal blood volume is an advisable and safe volume to give If active blood loss is present, the estimated blood loss should be replaced and can safely vary from minus 10% to plus 10% Since these estimates can vary by as much as 25% in either direction, it is safer to underestimate and then give more blood postoperatively if indicated, than to overestimate and then have to treat the complications associated with overtransfusion

REFERENCES

1 Leigh M D and Belton, M K *Pediatric Anesthesia* New York, Macmillan 1949
2 Gross R E *The Surgery of Infancy and Childhood* Philadelphia, Saunders 1953
3 Stephen C R *Elements of Pediatric Anesthesia* Springfield, Thomas 1954
4 Faulconer, A Jr and Latterell K E Tensions of oxygen and ether vapor during use of the semi-open air ether method of anesthesia *Anesthesiology, 10* 247 259 1949
5 Smith, C A *The Physiology of the Newborn Infant* 2nd Ed Springfield Thomas, 1951
6 Papper E M Renal function during general anesthesia and operation *J.A M.A* 152 1686-1689, 1953

thoracic These patients do not usually need any supplemental fluids since mild shifts and water deficits will be tolerated and compensated for rapidly and safely This leaves the seriously ill children and those who need extensive intra abdominal or intrathoracic surgery to consider If dehydration is present preoperatively, attempts should be made to remedy it before anesthesia is begun

The total amount of fluids required varies from about 50 cc per pound of body weight per 24 hours in the newborn to 20 cc in the older child (Figure 12) Of this, no

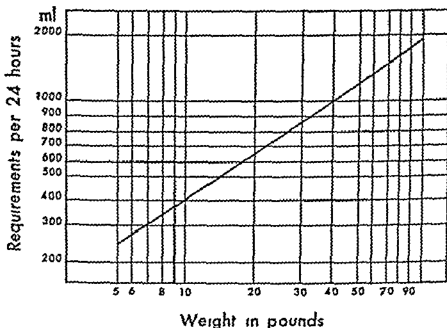


Figure 12 Fluid Requirements in Infancy and Childhood (From *Gross R Surgery of Infancy and Childhood* W B Saunders Co 1953)

more than 15% should be saline, preferably of one half isotonic strength The best intravenous fluid is 5% or 10% glucose in water[3] If it is given as a continuous infusion about 0 5 cc (8 drops) per minute is recommended for a 5 pound infant, 1 cc (16 drops) per minute for a 10-

It is because of this need to liberate the base by the normally alkaline tissue fluid that it is difficult to produce local anesthesia in acutely infected areas where the drug encounters an acid reaction in the tissues

The duration of action of the local anesthetics is proportional to the time during which they are in actual contact with nervous tissue Consequently procedures which localize the drug at the nerve greatly prolong the period of anesthesia Cocaine for instance constricts blood vessels and therefore delays its absorption For this reason the duration of cocaine anesthesia is greater than that of many of the other anesthetics which do not cause vasoconstriction The addition of a vasoconstrictor, the best of which is epinephrine, to local anesthetics prolongs and intensifies their action A concentration of 1 200,000 is sufficient for this purpose

Epinephrine performs a dual service By decreasing the rate of absorption it not only localizes the anesthetic at a desired site, but minimizes the possibility of toxic reaction due to absorption of large quantities of drug into the general circulation

I DRUGS

Certain properties are desirable in a good local anesthetic agent

First, it should have a selective action which is confined principally to nerve tissue

Second, it should not be irritating to adjacent structures upon local application

Third its action should be reversible

Fourth the systemic toxicity of a local anesthetic should be low because the componds are absorbed from their site of local application

Fifth the duration of action must be sufficiently long

XVI

LOCAL ANESTHESIA

LOCAL ANESTHETIC agents are drugs which are capable of blocking nerve conduction when applied locally to nerve tissue in effective concentration They exert this effect on any portion of the nervous system and on every type of nerve fiber The greater practical value of the local anesthetic drugs is due to the fact that their action is reversible and is followed by complete recovery of nerve function without evidence of structural damage to nerve fibers or cells

The intelligent and safe use of local anesthetic agents requires a thorough knowledge of their pharmacological actions and the prevention and control of toxic reactions

The local anesthetics are generally marketed in the form of their water soluble salts, usually hydrochlorides There is evidence to show, however, that it is the free base which acts on nerve tissue, for it is in their basic form that local anesthetics are lipoid soluble and lipoid solubility appears to be a necessary prerequisite for activity of any anesthetic agent whether local or general

It has been demonstrated that the addition of alkali to local anesthetic solutions increased to some extent their anesthetic potency However, after alkalinization the solution soon becomes turbid and the free base separates out Therefore as a practical measure alkalinization is not employed Rather one relies on the ability of the slightly alkaline tissue to liberate the active base

of its length of action It gives anesthesia for 1½ to 2 hours and with epinephrine lasts for 5 hours It also maintains analgesia for 9 or more hours aiding in the establishment of a comfortable postoperative period for the patient This length of action is an important feature in anesthesia for surgery of the toes and fingers where a digital block is used and epinephrine may be contraindicated By using this drug, long anesthesia without the use of epinephrine may be obtained

Xylocaine is a relatively new drug that was developed in Sweden Since its introduction into this country a few years ago, it has become very popular Although its toxicity has been reported to be low, a high proportion of reactions, especially with the 2% solution, have been seen in some instances Drowsiness may also be noted

Cyclaine is the most recent local anesthetic drug It has a low toxicity, quick onset, and lasts about 1½ times as long as procaine

II CONTRAINDICATIONS

The contraindications to local anesthesia are few It is not wise to inject an infected area both because there may be a mechanical spread of the infection and because no anesthesia may be obtained If an infected area is to be operated on, it is wiser to do a nerve block at a distance from the infection, rather than infiltrate locally The same applies to the presence of a dermatitis where the possibility of a secondary bacterial infection is present

It has been suggested by some that the use of local anesthetics should be avoided in patients with severe liver or kidney damage since these organs are important in destroying and eliminating these drugs

Extreme care should be taken with patients who give a history of having had a previous untoward reaction to a

to allow ample time for the performance of the contem plated surgical procedure

The time required for the onset of anesthesia is like wise important and should be as short as possible

Several of the more popular chemicals possessing these qualifications will be considered (Table IV) In discussing toxicity procaine will be designated as the standard equal to one

TABLE IV

Drug	*Toxicity (Procaine = 1)*	*Concentration Used (%)*	*Length of Action (min)* $\bar{s}$ *Epinephrine*	*Length of Action (min)* $\bar{c}$ *Epinephrine*
Procaine (Novocaine)	1	0 5–2	30–45	60–90
Metycaine	2–3	0 5–2	30–45	60–90
Pontocaine	12	0 1–0 15	90–120	300+
Xylocaine (Lidocaine)	1½	0 5–2	60–90	180–210
Cyclaine	1	0 5–1	45–90	75–120

Procaine (Novocaine®) is the most popular local an esthetic It is the least toxic of all these drugs It is used in concentrations of 0 5 to 2 0%, the lower concentration is adequate for infiltration anesthesia Two per cent usu ally is needed to obtain solid anesthesia when blocking nerves Its length of action without epinephrine is 30 to 45 minutes and with the vasopressor 60 to 90 minutes

Metycaine is somewhat more toxic than procaine It is used in the same concentrations and has a duration of action somewhat longer than procaine This drug is widely used for caudal anesthesia during obstetrical delivery However, its application to infiltration and other forms of conduction anesthesia is also very satisfactory

Pontocaine® (tetracaine) is 12 times more toxic than procaine but only one tenth the concentration is used This drug is a favorite in nerve block anesthesia because

The local anesthetics may also act adversely on the circulatory system Sometimes a rise in blood pressure is noted, but usually a fall occurs If the fall in blood pressure is marked it is wise to administer a vasopressor intravenously Ephedrine sulphate in doses of 15 to 25 mgms has proved effective Inhalation of 100% oxygen is advisable

On rare occasions, there may be syncope and a complete cardiovascular collapse following even the administration of relatively small amounts of the drug This reaction has frequently been termed an idiosyncrasy, but is also seen following the inadvertent injection of the drug intravenously Instantaneous artificial respiration with oxygen is imperative if the patient is to be saved, and even with immediate attempts at resuscitation, the collapse may be so sudden and so severe that death quickly ensues

The injection of these anesthetic agents is not the harmless, safe procedure that many people so frequently believe it to be It should be approached with caution and every precaution should be taken to avoid complications

After the needle has been inserted through the intradermal wheal and before drug is injected any place beneath the cutaneous layer, aspiration should be attempted in at least 2 planes to be sure that the needle tip is not lying within a blood vessel Aspiration in at least 2 planes is important because the bevel of the needle may be lying against the wall of a vessel and no blood will appear in the syringe when the aspiration is attempted since the vessel wall acts as a valve It is best to rotate the needle a full 180 for the second attempt at aspiration This procedure should be carried out every time the needle is moved or the syringe changed

The solution employed should be *doubly checked* to

local injection of an anesthetic, or who may give a pro found allergic history Skin testing is not a reliable means of preventing anaphylactic reactions

High blood pressure, hyperthyroidism, heart disease, arteriosclerosis and other peripheral vascular diseases contraindicate the use of epinephrine

III COMPLICATIONS

The toxic reactions and complications of all the local anesthetic drugs are referable to either the central nervous system or the cardiovascular system

The symptoms of nervous system dysfunction result from central stimulation and consist chiefly of restlessness, tremors, excitement, and sometimes nausea and vomiting These can proceed to generalized clonic convulsions The stimulation is then followed by depression, coma and death

The logical drugs to use in the treatment of poisoning by local anesthetics are the hypnotics and of this group the barbiturates are most valuable A fast acting barbiturate is given intravenously until the symptoms disappear Very effective drugs are thiopental and pentobarbital

No individual should undertake the injection of a local anesthetic without having an intravenous barbiturate readily available If convulsions or coma occur, artificial respiration with 100% oxygen is imperative in addition to the barbiturate to combat the marked cerebral hypoxia associated with these conditions

If a patient becomes apprehensive and trembles a little during the injection of a local anesthetic this should not be discounted as being due to nervousness or hysteria on the part of the patient It may be due to the drug and it is wiser to discontinue injecting and administer a small dose of barbiturate before continuing

XVII

SPINAL ANESTHESIA

THE ADVANTAGES of spinal anesthesia include profound muscular relaxation, contraction of the gut, non explosive technique, minimal disturbance of acid base balance, employment of simple portable equipment (syringes, needles) and the relatively simple procedure of lumbar puncture It is necessary to have at hand means for ventilation with oxygen and vasopressor agents for intravenous use to prevent and correct hypotension

The site of injection is usually midline, between L-2 and L-5 interspaces If the interspinous ligaments are calcified the paramedian approach is useful The lateral paravertebral approach may also be employed

The level of spinal anesthesia is of importance and varies with the operative procedure contemplated It is desirable for the anesthesiologist to keep a level of anesthesia as low as possible and still suitable for the opera tion proposed since the disturbances of circulatory and respiratory physiology are of greater frequency and magnitude as the level ascends Level depends on a number of factors

1 *The Volume of Solution Injected* The greater the volume, the higher the level of block

2 *Position of the Patient During and Immediately Following the Injection* Hyperbaric solutions ascend to higher levels by placing the patient in Trendelenburg position Once the desired level is reached the table

be sure it is the correct solution Unlabeled bottles should not be used Antiseptic solutions must never be confused with anesthetic solutions

The lowest concentration and smallest total dose of the drug consistent with successful anesthesia should be used A wise precaution is slow injection so that if a reaction does occur a smaller dose will have been administered

IV HYALURONIDASE

Hyaluronidase (the so called spreading factor) is an enzyme which promotes diffusion in the tissues If added to the local anesthetic solution, it increases the skin area anesthetized and causes a faster onset of anesthesia However, due to this diffusion the duration of anesthesia is reduced It is not particularly useful as an aid to local anesthesia

pensatory vasoconstriction in the unanesthetized zone has been demonstrated [2]

The heart rate will slow if high thoracic levels are reached This is probably due to paralysis of the sympathetic cardioaccelerator fibers permitting a greater vagal effect on the heart

Respiration Respiratory depression is directly associated with the height of motor block As the spinal level ascends the intercostals are paralyzed If cervical levels are reached, phrenic nerve paralysis will cause a cessation of respiration Immediate therapy by artificial respiration with 100% oxygen is imperative When a total spinal block is produced the patient will lose consciousness even though adequate blood pressure levels are maintained and artificial respiration has been instituted A possible cause of coma is a loss of all sensory impulses to the cortex which induces sleep

Gastro intestinal Tract Sympathetic paralysis causes over-activity of the parasympathetic nerves with resulting contraction of the gut This is of advantage to the surgeon

Nausea and vomiting occur frequently, especially with high levels and low blood pressure Relief is frequent with inhalation of 100% oxygen and light general narcosis

COMPLICATIONS

Neurological

1 Headache This is the most common postoperative complication It occurs in 4 to 14% of patients [3, 4] The cause is believed to be hypotension of the cerebrospinal fluid secondary to leakage through the site of dural puncture [4] The number of headaches has been remarkably reduced by the use of a 22 gauge or smaller needle At-

should be placed horizontally The anesthesia will usually ascend further for one or two more segments It is advisable to keep the head well flexed at all times to prevent anesthesia of the cervical segments which will cause respiratory paralysis For sacral anesthesia, the sitting position may be used for the spinal tap and maintained for a few minutes after the solution is injected to confine the anesthesia to the sacral nerve roots

3 *Rate of Injection* The faster the drug is injected the higher the level

4 *Vertebral Level of Injection* This has minimal effect on the height of anesthesia The higher the point of injection, the higher the block

5 *The Amount of Drug Injected* The larger the dose, the higher the level, other factors remaining constant

PHYSIOLOGICAL CHANGES FOLLOWING SPINAL ANESTHESIA

Circulation Arterial blood pressure will fall, especially if the spinal anesthesia is relatively high A vasopressor (ephedrine, methedrine, neosynephrine) should be given intramuscularly just prior to spinal tap to avoid this complication If hypotension occurs after the spinal has been given, the vasopressor must be injected intravenously

The cause of the hypotension is a loss of vasomotor tone in the arterioles and pooling of blood in the post arteriolar bed (capillaries and veins) proportionate to the *degree of sympathetic paralysis* [1] *This results not only in a* decreased peripheral resistance but also in a decreased venous pressure, diminished return of blood to the right heart and lowered diastolic volume which decreases the stroke volume and cardiac output In lower level spinal anesthesia sympathetic activity is partially intact, com-

2 Injury to Nervous Structures If the lumbar puncture is at or below the L2 3 interspace, the cord will not be damaged If a nerve root is hit, the needle should be moved so that the paresthesia is gone before the anesthetic agent is injected

3 Bleeding If a bloody tap is performed which does not clear readily, another interspace should be tapped before introducing the anesthetic agent

CONTRAINDICATIONS TO SPINAL ANESTHESIA

1 Any known central nervous system or nerve root disease

2 Syphilis, especially if there is central nervous system involvement

3 Motor disturbances of the bladder or rectum

4 Anticoagulant therapy

5 A previous history of delayed restoration of motor functions or severe paresthesias following a previous spinal anesthesia

6 Hemorrhagic or traumatic shock or seriously reduced blood volumes

AGENTS—HYPERBARIC SOLUTIONS

1 Procaine crystals, dissolved in spinal fluid or normal saline 3 to 5% concentration Dose 50 to 150 mgms

2 Pontocaine, weighted with 5 0 to 7 5% dextrose solution, 0 3 to 0 5% concentration, dose 5 to 15 mgms

3 Nupercaine, in 0 25% concentration with dextrose, dose 2 5 to 7 5 mgms

Hypobaric Solutions

1 Nupercaine 1 1500 (0 67 mgm /cc) in 0 5% saline solution (Jones solution), dose 8 to 20 cc

2 Pontocaine 1 1000 (1 mgm /cc) in distilled water, dose 5 to 15 cc

tention should also be paid to the bevel of the needle, inserting it so that it faces laterally and will not cut the longitudinal dural fibers Adequate hydration of the pa tient will also help diminish the incidence of headaches

Therapy consists of bed rest, hydration, and the injec tion of saline into the epidural space

2 ABDUCENS NERVE PARALYSIS WITH DIPLOPIA This is a rare complication and always clears up The cause is probably the same as that for headache

3 SEPTIC MENINGITIS This is caused by a break in sterile technique or lumbar puncture through an infected area Prevention is obvious Spinal anesthesia is contra-indicated for patients with highly virulent infections (bacteremia), nephrostomy and colostomy, and skin infection at the site of lumbar puncture

4 ASEPTIC MENINGITIS, ARACHNOIDITIS, RADICULITIS These more serious complications are rare They may result from the injection of too concentrated a solution, injection of the wrong drug (alcohol), leakage into the anesthetic ampules of antiseptic solutions, or detergents in syringes These vary in intensity from meningeal irrita tion to a cauda equina syndrome with paralysis and loss of sensation of the lower extremities and urinary and rectal incontinence

Complications of Lumbar Puncture These occur after lumbar puncture alone and are not necessarily due to the anesthetic A number of structures must be pierced be fore the subarachnoid space can be reached by the nee dle If a sharp small gauge needle is introduced expertly trauma will be minimal If a dull large bore needle is used and if multiple explorations are required the extent and degree of injury are increased

1 BACKACHE This is the most frequent complaint besides headaches

2 Injury to Nervous Structures If the lumbar puncture is at or below the L2-3 interspace, the cord will not be damaged If a nerve root is hit, the needle should be moved so that the paresthesia is gone before the anesthetic agent is injected

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REFERENCES

1 Papper, E M, Bradley, S E and Rovenstine, E A Circulatory adjustments during high spinal anesthesia *J A M A*, *121* 27 32, 1943

2 Neuman, C, Foster, A D and Rovenstine, E A Importance of compensatory vasoconstriction in unanesthetized areas in maintenance of blood pressure *J Clin Investigation* *24* 345-351, 1945

3 Dripps R D and Vandam, L D Long term follow up of patients who received 10,098 spinal anesthetics *J A M A*, *156* 1486-1491, 1954

4 Thorsen, G Neurological complications after spinal anesthesia *Acta chir scandinav*, 95 Supp 121, 1947

XVIII

CAUDAL AND EPIDURAL ANESTHESIA

THE EPIDURAL space extends from the foramen magnum to the ligament covering the sacral hiatus It is limited anteriorly by the dura and extends laterally to the vertebrae and through the lateral intervertebral spaces There is no epidural space in the skull as the dura attaches to the foramen magnum The "space" is the site of some degree of negative pressure, related to the negative thoracic pressures of inspiration This is not enough to be transmitted to the sacrum

Anesthetic solutions can be deposited anywhere in the epidural space and will cause anesthesia of the mixed nerve roots which traverse the space Recent studies also indicate that some drug will reach the subarachnoid space and cause dorsal root ganglion blockade[1] The number involved depends on the volume of solution injected, its concentration and the site of puncture Since no spinal fluid is present to dilute and diffuse the solutions, weaker concentrations of greater volume than those used for spinal anesthesia, will produce the desired results The injections should be made at those sites where the epidural space is the largest midcervical, midthoracic, lumbar below L-2 and into the sacral canal, to minimize the serious complication of puncturing the subarachnoid space The sites of the cervical and lumbar enlargements of the cord are accompanied by the smallest area of epidural space

Advantages A nonexplosive method is available which requires relatively inexpensive and simple equipment The drugs used cause minimal metabolic changes even in very ill patients Catheter methods are frequently used, allowing for prolonged administration of drugs

Technique Dexterity with needles and catheters is necessary for success The epidural space is recognized in two ways One, by sensing the resistance of the sub flavian ligament as the needle traverses it, attaching a small syringe with 1 to 2 cc of solution and noting that the plunger is difficult to advance, insertion of the needle 1 to 2 mm deeper allows the plunger to be depressed easily Aspiration with the plunger shows that no spinal fluid can be withdrawn The conclusion can then be reached that the epidural space has been entered The drug can then be introduced or the catheter inserted through the needle An alternative method utilizes the negative pressure in the epidural space The stylus of the needle is removed when the subflavian ligament has been reached and a drop of fluid inserted into the hub of the needle It is then advanced slowly The drop disappears into the hub of the needle when the epidural space is reached The failure to aspirate spinal fluid again signifies that the epidural space has been entered

Caudal (a special form of peridural) block is accom plished by the insertion of a needle into the ligament covering the sacral hiatus, and advancing it between the outer layer of the sacrum formed by the laminae and spinous processes, and the inner layer formed by the bodies of the sacral vertebrae In most patients, the dura tapers to a point ending at the S 2 segment, and spinal fluid may occasionally be obtained from this site, especially in short patients It is necessary to recognize dura puncture for if the larger volume of dilute drug usually used for epi-

dural anesthesia is inadvertently injected into the subarachnoid space a high spinal block and death may ensue

The resultant circulatory collapse and respiratory paralysis must be treated promptly with artificial respiration with oxygen and circulatory support For this reason, a test dose of a small volume of anesthetic solution is always administered before the usual dose is given

It is apparent that the technique is more time consuming than spinal anesthesia and the anesthesia has a slower onset which is often a disadvantage On the other hand, headaches and other neurological sequelae are infrequent if spinal puncture is avoided

Drugs and solutions in common usage are 1 to 2% procaine, 1 5% metycaine, 0 15% pontocaine and 0 75% to 2 0% xylocaine Epinephrine in concentration of 1 200,000 added to any of these will prolong the duration of anesthesia The usual single dose is 20 to 30 cc With the continuous catheter technique increments of 10 cc are added as needed

REFERENCE

1 Frumin, M J *et al* The appearance of procaine in the spinal fluid during peridural block in man *J Pharmacol & Exper Therap* 109 102 105 1953

XIX

OBSTETRICAL ANESTHESIA

THE AIMS of obstetrical anesthesia are pain relief to the mother with no added risk to the mother or baby, and with no interference with the course of labor These ideals are not completely achieved in actual practice

All forms of pain relief apparently diminish the force of uterine contractions to some extent However, the transformation of a restless unmanageable patient into a willing cooperative subject able to exert abdominal pressure at the correct time (by a successful regional block) more than makes up for the loss of power of uterine contractions

If the block methods are performed properly, and maternal hypotension and convulsive manifestations of drug overdosage are avoided, the risk to the infant is slight, as compared with the usual general anesthesia preceded by narcotic drugs for the first stage of labor However, caudal epidural and spinal anesthesia are somewhat more difficult to administer in the full term obstetrical patient than the surgical one Sacral edema and pelvic congestion are frequent and good spinal flexion is not easy to obtain in the full term pregnant woman For these reasons many types of general anesthesia are still in use

In general, regional anesthesia is favored if the infant is premature, if it has fetal distress, if moderate maternal bleeding has occurred, or if operative obstetrics is indicated The use of regional anesthesia increases the need

for forceps extractions by increasing the incidence of persistent posterior positions Regional anesthesia is not indicated in cases of uterine inertia or cephalopelvic disproportion for maximum uterine power is desirable There is no good evidence that cervical dystocia is aided by regional anesthetic methods though they are often used in an attempt to relax the cervix Cesarean section during spinal anesthesia is favored because of the relative lack of effect on the infant A dose of vasopressor drug and an intravenous infusion started before the lumbar puncture is performed are mandatory to reduce the incidence of hypotension and to insure an easy quick route of treatment of hypotensive episodes When general anesthesia is used for Cesarean sections an attempt is made to produce as little respiratory depression in the infant as possible Accordingly the patient is prepared and draped and the surgeon ready to make the incision before the anesthetic administration is begun Induction with a fast acting agent such as cyclopropane followed by quick surgery will frequently result in delivery of an infant breathing spontaneously

Two very important obstetrical complications are handled primarily by the anesthesiologist, hemorrhage and aspiration of vomitus Obstetrical hemorrhage can be sudden, dramatic, and fatal It is the responsibility of the anesthesiologist to start intravenous fluids through large bore needles, use plasma expanders until blood is available, and keep ahead of the blood loss if at all possible

The full stomach of the multipara who is admitted in rapid labor has been the chief cause of maternal mortality, but it is now relegated to third place Preservation of the patient s own laryngeal and pharyngeal reflexes is important so that pudendal block saddle spinal or caudal anesthesia are often used in this group

Every patient in labor should be considered individually by the anesthesiologist in his selection of pain relieving drugs, in consultation with the obstetrician who will inform him as to obstetrical problems A plan of pain relief can then be formulated, which may be subject to change at very short notice depending upon the progress of labor and the condition of the infant

NEWBORN INFANT RESUSCITATION

Newborn infant resuscitation frequently becomes the responsibility of the anesthesiologist, especially at times when the obstetrician is properly busy with maternal obstetrical problems, and the pediatrician has not yet been summoned The situations of respiratory obstruction, apnea and ventilation are familiar to the anesthesiologist, and if the maternal problems are under control he may assume direction of infant resuscitation All three groups anesthesiologists, obstetricians, and pediatricians should be familiar with such problems

The establishment of a free airway is fundamental in any resuscitative problem The cilia of an oxygenated infant can easily remove amniotic fluid which is always present in the tracheobronchial tree Meconium and blood from the birth canal are more difficult to remove Rapid, gentle pharyngeal aspiration will remove these materials grossly It is unlikely that either bronchoscopy or pulmonary lavage will remove enough from the deeper bronchi to justify their use

All infants are cyanotic at birth, since the saturation of arterial blood is between 50 and 70% However, adequate spontaneous respiration within the first 2 minutes after birth, usually brings this saturation up to 95% If the infant breathes actively but minute volume exchange is poor it is desirable to add an oxygen enriched atmos-

phere for cerebral damage can be prevented if oxygenation is maintained, even if hypopnea persists for many minutes If *no* respiratory efforts are present (*apnea*) it is desirable to inflate the infant's lungs for brief periods to some degree of positive pressure Inflations with a face mask are indicated although distension of the stomach in preference to the lungs may occur However, if improvement of the slow heart rate does not occur in a few seconds, direct inspection of the infant s larynx is indicated and foreign material aspirated if present followed by the insertion of an endotracheal tube which is simple in a flaccid, apneic patient and insures direct inflation of the lungs with oxygen One or two brief inflations insure the best expansion with the least damage

Several operable anomalies may be diagnosed at birth The stomach of all infants should be entered with a rub ber catheter introduced into the pharynx in the midline If it cannot be advanced easily, blind esophageal pouch should be suspected, and the diagnosis of tracheoesophageal fistula is made until ruled out The history of hydramnios in the mother suggests other anomalies as well such as duodenal atresia and especially diaphragmatic hernia, which is a frequent cause of persistent or increasing cyanosis and is usually fatal unless corrected surgically in a few hours

Pulmonary expansion is effected better if the liver pulls the diaphragm downward, the head up position is desirable and preferable to the head down position as soon as a free airway has been established

Heated blankets and cribs have been in vogue for many years, but it is probable that cooling of the infant is more physiological and will reduce his need for oxygen, especially if some interference with oxygenation is present

A system of "scoring of newborn infants is in use at

present and includes observations of respiratory effort, heart rate, muscle tone, reflex irritability and color sixty seconds after the cord is clamped (Table V)

TABLE V

Score	*0*	*1*	*2*
Heart rate	None	<100	>100
Muscular tone	None	Poor	Good
Respiratory effort	None	Inadequate	Crying
Reflex irritability (Response to nasal catheter)	None	Grimaces	Sneezes or coughs
Color	All cyanotic	Hands and feet cyanotic	All pink

Anesthetic and resuscitative efforts may be evaluated from this system Scores of 8 9 or 10 are excellent

XX

RESUSCITATION

RESUSCITATION refers to the restoration of respiration and circulation in those subjects in whom one or both of these vital functions has become markedly depressed or ceased

If one considers that it is only a matter of minutes before irreparable damage occurs to the brain cells during total anoxia which ensues following cessation of respiration or cardiac arrest, it will become obvious that only prompt intervention according to a pre set plan can prevent fatalities

Respiratory and circulatory resuscitation will be discussed separately but it must be stressed that these must be carried out simultaneously espeically when cardiac arrest occurs

RESPIRATORY RESUSCITATION

If a patient has stopped breathing or respiratory movements are insufficient, artificial respiration must be instituted immediately Artificial respiration refers to a method of maintaining normal physiological tensions of oxygen and carbon dioxide in the alveoli

Before resuscitation is begun the operator must be certain that there is no obstruction to the passage of gases to and from the alveoli Vomitus, mucus, and foreign bodies must be removed If the tongue obstructs the airway, the jaw should be held forward and an oropharyngeal or nasopharyngeal airway inserted The latter may be indicated if the mouth cannot be opened

Respiratory resuscitation of varying degrees of effectiveness can be accomplished in one of two ways

I) Movement of gases can be produced by a force applied to the exterior of the thorax Examples of this are

1 Manual Methods

a SCHAEFFER METHOD[1] The patient is placed prone with the head turned to the side and resting on one arm The operator straddles the patient and exerts pressure with his palms placed below the last rib to compress the thorax Quick release will allow the tissues to recoil and produce inspiration Only about 200 cc of exchange can be accomplished by this method This method is inefficient and is no longer recommended

b EVE METHOD[2] The patient is secured on a board in the prone position The board is placed on a fulcrum and seesawed head up then head down about 30 degrees in each direction This method allows about 250 cc exchange so that it is about 50% efficient

c SILVESTER METHOD[1] The patient is placed in a supine position and the operator kneels at the head The arms of the patient are then raised over the head to produce inspiration and then lowered to the side and compressed across the chest to cause expiration About 500 cc of air are moved by this method

d HOLGER-NIELSEN METHOD[3] The patient is placed prone with arms overhead The head is turned to one side and rests on a hand The operator kneels on one knee above the head of the patient and places the foot of the other leg near the patient's elbow The hands are placed on the subject's back on a line with the axillae Pressure is exerted on the chest until the operator's arms are vertical Pressure is then released slowly, the subject's arms

are grasped above the elbows and raised until resistance is felt at the subject's shoulders The arms are then dropped and chest pressure resumed This is one of the most efficient methods and causes an exchange of about 580 cc of air

2 *Mechanical Methods* The Drinker respirator (iron lung) *where the body of the patient is placed within a* chamber while the head remains outside A motor drives a diaphragm which increases and decreases the pressure about the chest to cause expiration and inspiration respectively

II Expansion of the thorax can be produced by inflating gases under pressure into the lungs intermittently Examples of this are

1 ***Mouth to mouth*** breathing

2 ***Rubber mask and breathing bag*** such as that available on the usual anesthetic machine This is the method of choice in the operating room It is not only convenient but 100% oxygen can be used An endotracheal tube will greatly facilitate this form of resuscitation but it is not necessarily essential if time is wasted in obtaining and in serting the tube Artificial ventilation must be maintained without the tube until a convenient period for intubation presents itself Intermittent positive pressure on the bag produces the desired result It is important to remember that at least as much time must be allowed for expiration as inspiration An absorbing system for the removal of CO is also necessary

3 *Mechanical Devices* The Kreiselman resuscitator, the Kreiselman hand bellows,[4] the Rand respirator, the Jefferson ventilator and others

As stated earlier all these methods presuppose an unobstructed airway If the upper respiratory tract is ob

structed, however, a tracheostomy may be necessary to allow the oxygen to reach the alveoli

The rate of artificial respiration should be about 16 to 20 times per minute for all methods

CARDIAC RESUSCITATION

Cardiac arrest refers to two conditions of the heart which will bring the circulation to a halt

1 *Cardiac Standstill* The heart has stopped beating entirely

2 *Ventricular Fibrillation* The ventricles are quivering so fast and weakly that complete circulatory arrest is present

The diagnosis of cardiac arrest is made if no pulse is palpable nor blood pressure recordable Usually the patient also has ceased breathing No time should be wasted in listening to the chest for heart sounds It is better to open the chest and find the heart beating than to lose a patient by hesitating too long Three minutes is the limit for survival of the cerebral cortex without damage

An incision is made in the 4th or 5th intercostal space on the left and the thoracic cage entered The heart is then massaged at the rate of about 60 to 70 times a minute Cardiac massage properly done consists of squeezing the heart from the apex toward the base in a 'milking action

In cases of cardiac standstill where massage has been instituted promptly, the heart will frequently beat spontaneously and produce adequate blood pressures If after a few minutes of massage the heart does not start, remains soft and flabby, or begins to beat very weakly, certain drugs are given Calcium chloride (10%) in doses of 3 to 5 cc or epinephrine (1 10,000) 3 to 5 cc directly into the right ventricle of the heart are recommended

In cases of ventricular fibrillation cardiac resuscita-

tion is begun as above, with massage However, the fibrillation must be stopped before the heart can be expected to work efficiently again This can be accomplished by defibrillation with 100 to 110 volt alternating (60 cycle) electrical current having 1 0 to 1 5 amperes The current is passed through the myocardium by means of two electrodes placed directly on the heart About 0 5 second shocks are used three to five times, allowing about 2 seconds between each shock This will result in asystole which is treated again by massage and calcium chloride until the heart regains a forceful regular beat Defibrillation should not be started until the myocardium appears well oxygenated Epinephrine is avoided in cases of myo cardial anoxia The injection of procaine into a ventricle has been recommended for treatment of this condition but its value is questionable

REFERENCES

1 Gordon A S, *et al* Critical survey of manual artificial respiration *J A M A* *147* 1444-1453 1951
2 Eve F C Resuscitation of the drowned today *J A M A*, *124* 964 967 1944
3 Council on physical medicine and rehabilitation report Back pressure arm lift method for administering artificial respiration recommended *J A M.A*, *147* 1454 1455 1951
4 Kreiselman J A new resuscitation apparatus *Anesthesiology* 4 608 611, 1943

XXI

ANESTHETIC CONSIDERATIONS IN SURGICAL EMERGENCIES

In surgical emergencies preoperative preparation is fre quently incomplete through force of circumstances Fol lowing is a discussion of the main factors or problems which must be considered for emergency cases

1 *Fluid Therapy* Extracellular fluid deficit is com mon in these cases and should be replaced by appropriate intravenous solutions Acute blood loss should be re placed by blood If blood is not rapidly available, shock can be temporarily alleviated by plasma expanders (Dex- tran 500 to 1500 cc) given intravenously

2 *Premedication* The full effect of premedication is obtained by the *intramuscular route in about 30 minutes* If a more rapid effect is required the premedication is administered intravenously with about one half to two thirds of the intramuscular dose The presence of pain suggests larger premedicant doses whereas shock, tox- icity, and debilitation may indicate the use of smaller doses or omission of premedication Inquiry should always be made whether an injured patient has already received some narcotic for pain and the time of its administration so that overdosage will not occur

3 *The Full Stomach and Intestinal Obstruction* His- tory of a recent meal means that solids or liquids may be present in the stomach This is almost certain to be the case within four hours and possibly up to twelve hours

Vomiting is likely during induction of general anesthesia Fatal airway obstruction or subsequent aspiration bronchopneumonia are the potential consequences Emptying of the stomach through a gastric tube or by induced vomiting may leave an unevacuated residue behind In such circumstances relatively safe anesthesia may be provided by regional methods with light premedication—spinal, caudal epidural, or local field block If these are unsuitable, the patient is intubated awake with a cuffed endotracheal tube after topical anesthesia to the pharynx larynx and trachea The cuff is inflated before the induction of general anesthesia An alternative is the production of respiratory paralysis with a muscle relaxant and rapid intubation in the semi recumbent position Both methods require some skill and experience

4 *Acute Alcoholism* These patients frequently have full stomachs If the patient is too uncooperative for regional methods it is essential if at all possible to perform intubation before induction If this proves impossible, a quick induction with intravenous thiopental and a muscle relaxant drug followed by intubation with the patient in the Fowler position is the best alternative despite the risk of regurgitation This method is not recommended for the novice because dexterity and speed in performing the intubation are absolutely essential

SPECIFIC TECHNIQUES FOR SPECIFIC EMERGENCIES

1 *Bleeding Tonsillar Fossa* Children with this complication can be quickly induced with cyclopropane and intubated Swallowed blood increases the risk of vomiting so that suction should be readily available

2 *Fractured Mandible* The problem in these cases is the inaccessibility of the mouth for airway purposes

Therefore nasotracheal intubation in the conscious state is advisable The nostrils are sprayed with cocaine (4 10%) in sufficient volume to allow it to reach the pharynx A well lubricated nasotracheal tube is introduced and advanced by stages, spraying through the tube at each step, until the cords have been passed and the trachea entered before general anesthesia is administered If the nose is also injured, preliminary tracheostomy may be necessary

3 *Ludwig's Angina, Markedly Deviated Trachea, Trismus, and Ankylosis of the Jaw* In all these conditions the airway must be assured with an endotracheal tube, either oral or nasal, inserted with topical anesthesia before the patient can be safely anesthetized with general narcosis

4 *Bronchoscopy* for aspirated foreign bodies in children Deaths have occurred when bronchoscopy has been attempted during general anesthesia Therefore it is recommended that this procedure be performed with heavy medication and topical anesthesia while continuously insufflating oxygen through the bronchoscope

XXII

FIRES AND EXPLOSION HAZARDS

THIS COMPLICATION is very infrequent It occurs about once in every 250,000 anesthesias However, it is one of the most dreaded of all disasters and always secures a prominent place in the daily press

Of the common anesthetics used the following are flammable

Ether
Cyclopropane
Ethylene
Vinethene
Ethyl chloride

Nitrous oxide, chloroform and trichlorethylene are non flammable

Sources of ignition include chiefly

1 Flames cigarettes, etc
2 Electrical equipment which is not spark proof
3 Percussion sparks
*4 Static sparks—which are probably the greatest danger as a source of ignition Electrostatic charges can set up dangerous potential differences in the presence of electrically nonconducting materials which act as barriers to the free movement of such charges and hence prevent equalization of potential differences A spark discharge can take place only when there is no other path of greater conductivity available by which this equalization may be effected

* Star indicates most important source

The measures taken to prevent this hazard include

1 Conductive flooring in the operating and anesthesia induction rooms, which provides a path of moderate electrical conductivity between all persons and equipment making contact with the floor to prevent the accumulaion of dangerous electrostatic charges Maximum resist ance of the floor must be less than 500,000 ohms as meas ured between two electrodes (5 pounds in weight and 2½ inches in diameter) placed 3 feet apart at any point on the floor The resistance must furthermore be over 25,000 ohms to protect against electrical shock

2 Conductive equipment properly grounded to the floor This includes machines, electrical equipment, and furniture in the operating room

3 All persons entering the anesthetic area must

a Wear conductive shoes Resistance between the inside and the soles must not be over 250,000 ohms Shoes should be tested before entering a room where explosive anesthetic agents are being used

b Not wear as part of their outer garment any material made of either wool, silk, nylon, sharkskin, or rayon No woolen blankets should be present

4 Electrical equipment and wall sockets are spark proof Non explosion proof electrical wiring and fixed equipment must be installed at least 5 feet above the floor in rooms used as anesthetizing locations

5 Relative humidity is maintained at an optimum of 55 to 60

6 When cautery is necessary for the operative proced ure no explosive agent is used Illuminating equipment which contacts patients (proctoscope and bronchoscope) is safe with flammable agents if an E M F of 8 volts or less is used for this equipment

7 When an anesthetic is given at a site where no con-

ductive precautions are present, a nonflammable technique must be used

8 Residual ether in vaporizers should be disposed of and never left overnight because ether peroxides formed by the oxidation of the ether over a period of time may cause an explosion in the anesthetic machine

9 Cylinders with flammable gases and cans of flammable liquids are stored away from cylinders of oxidizing gases (O_2 N_2O) in separate rooms

It is the responsibility of the anesthesiologist to help prevent anesthetic fires or explosions by seeing to it that all the rules are followed not only by himself but by all other personnel present

LIMITS OF FLAMMABILITY OF GASES AND VAPORS

In Air

4% 14 8% Ethyl chloride
3% 28 6% Ethylene
2 4% 10 3% Cyclopropane
1 8% Ether 48%
1 7% 27% Vinethene

In 100% O_2

2 9% Ethylene 79 9%
2 48% Cyclopropane 60%
2 1% Ether 82%
1 8% Vinethene 85%

Figure 13

REFERENCE

1 National Fire Protection Association *Recommended Safe Practice for Hospital Operating Rooms* Boston, 1953

XXIII

TABLES OF NORMAL VALUES

CIRCULATION

Age	*Pulse Rate (per min.)*	*Age*	*Blood Pressure Systolic*	*Diastolic*
Newborn	140-180	Newborn	70-80	30-40
1 week	124-130	1 week	80-90	40-50
3-6 months	130-140	3-12 months	85-95	45-55
6-12 months	110-130	1-8 years	90-100	50-60
1-5 years	100-120	8-12 years	100-120	60-80
5-8 years	90-100	Adult	110-140	70-80
8-12 years	80-90			
Adult	60-80			

Blood Volume—85 cc. per kilogram of body weight.
Plasma Volume—45 cc. per kilogram of body weight.
Red Cell Volume—40 cc. per kilogram of body weight.

Correction Factors for Blood Volume

1. Sex—subtract 5% for females.
2. Age—a. Subtract 10% if 50-65
   b. Subtract 15% if over 65
3. Obesity—Add ⅓ of the difference between the ideal weight and the obese weight.
4. Weight loss—
   a. If it has occurred within the last 2 months, use the original weight.
   b. If it occurred 2 to 6 months previously, use the present weight plus ½ of the weight lost.
   c. If it occurred 6 to 12 months ago, use the present weight plus ¼ of the weight lost.
   d. If it occurred over 12 months ago, use present weight.

Concentrations of Electrolytes in the Plasma

Sodium —	142 meq/L	Bicarbonate —	27 meq/L
Potassium —	5 meq/L	Chlorides —	102 meq/L
Calcium —	5 meq/L	Phosphates —	2 meq/L
Magnesium —	3 meq/L	Sulfates —	1 meq/L
		Organic acids —	6 meq/L
		Proteins —	16 meq/L

Other Plasma Constituents

Glucose —	70 100 mgms /100 cc
Non protein nitrogen —	25-35 mgms /100 cc
Urea nitrogen —	10 15 mgms /100 cc
Cholesterol —	150 190 mgms /100 cc

Contents of Blood Bottles (ACD—Solution)

(Total volume = 120 cc)		*Total for 120 cc*
Dextrose	1 40 gm %	1 68 Gm
Sodium citrate	1 32 gm %	1 58 Gm
Citric acid	0 48 gm %	0 58 Gm

RESPIRATION

Age	*Respiratory Rate (per min)*	*Tidal Volume (cc)*
Newborn premature	58	12
Full term	40 50	20
3 months	45	25
6 months	40	35
1 year	35	60
3 years	30	100
6 years	27	150
11 years	24	200
14 years	22	250
Adult	16	450 500

XXIII

TABLES OF NORMAL VALUES

CIRCULATION

Age	*Pulse Rate (per min)*	Age	*Blood Pressure Systolic*	*Diastolic*
Newborn	140 180	Newborn	70 80	30-40
1 week	124 130	1 week	80 90	40 50
3 6 months	130 140	3 12 months	85 95	45 55
6 12 months	110 130	1 8 years	90 100	50 60
1 5 years	100 120	8 12 years	100 120	60 80
5 8 years	90 100	Adult	110 140	70 80
8 12 years	80 90			
Adult	60 80			

Blood Volume—85 cc per kilogram of body weight
Plasma Volume—45 cc per kilogram of body weight
Red Cell Volume—40 cc per kilogram of body weight

Correction Factors for Blood Volume

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2 Age—a Subtract 10% if 50 65
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c If it occurred 6 to 12 months ago use the present weight plus ¼ of the weight lost
d If it occurred over 12 months ago use present weight

INDEX

ANATOMICAL DISTANCES (AVERAGE)

Age	Lips to Carina (cm)	Length of Larynx (cm)	Length of Trachea (cm)
Birth	12	1 7	4 0
1 year	14		4 5
2 years	15	2 0	5 0
5 years	17	2 4	5 5
10 years	18	2 5	6 5
15 years	23	3 0	7 5
Adult	26 29	4 0 5 0	10 15

AVERAGE DIAMETER OF GLOTTIS

Age	(mm)
Birth	5 6
1 year	6 7
2 years	7 8
5 years	8 9
10 years	9 10
15 years	10 11
Adult	11 14

This Book

MANUAL OF ANESTHESIOLOGY

By

HERMAN SCHWARTZ M D,
S H NGAI, M D
E M PAPPER, M D

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